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"A STUDY OF MALTA FEVER"

with Cases.

Being a Thesis for the Degree of M.D.,  
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by

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## I N T R O D U C T I O N .

Although much advance has been made in the study of Malta Fever during the past few years, the average medical practitioner in this country has, even yet, only a hazy idea of the nature of the disease. This is not to be wondered at considering that the disease is practically non-existent in the British Isles, and is only now beginning to be thoroughly understood in the sub-tropical zone where it flourishes.

The writer was led to make a more complete study of this fever, through having himself fallen a victim to it. As a result of it, he very fully realised how important it was that those in charge of such a case in this country should be thoroughly conversant with its diagnosis and treatment .

Before taking up my own and other cases, I shall endeavour to give a general description of Malta Fever, so far as our present knowledge of the disease will allow us.

### Short History of Malta Fever:

This fever was known to exist in the beginning of the nineteenth century, and it probably also occurred in the eighteenth century and even further back.

It was recognised as a variety of continued fever, but was constantly being confused with



enteric and malarial fevers. Burnett in 1810 described certain cases of Mediterranean Fever which he had met with, but as many of these were of short duration and had a high mortality, no doubt a large number of them were cases of Enteric Fever.

Marston in 1859 wrote the first description of the fever as a distinct disease, when he gave a very clear and complete clinical picture of it.

Neale in 1879 was the next to contribute anything of value to our knowledge, when he described cases of the fever invalided to Netley from Gibraltar and Malta.

An important year was 1887 when D. Bruce discovered the specific organism of the disease which he named *Micrococcus Melitensis*. In 1897 A. E. Wright and Temple applied the method of serum diagnosis to this fever, which has been of inestimable value in assisting to work out the subject and to differentiate it from enteric, malarial and other acute specific fevers.

The same year also saw the publication of the most complete study of the fever, when "Undulant, Malta or Mediterranean Fever" by Surg. Capt. M. Louis Hughes appeared.

Much original work has been done since then, more particularly with regard to Etiology, and treatment by antitoxins.

Much sound work has also been done by the Commission appointed by the Admiralty, the War Office and the Civil Government of Malta, for the investigation of Malta Fever. The reports of this commission were published in 1905.

#### Synonyms:

The number of names given to this fever is innumerable.

The following are the better known ones:-

Malta Fever: Mediterranean Fever: Undulant Fever:  
Rock Fever: Country Fever (Constantinople) Italian  
or Neapolitan Fever (in Candia): La Febbre Gastro-  
biliosa: Faeco-malarial Fever: etc., etc.

#### Definition:

A non-contagious endemic fever with undulations of pyrexia, separated by short periods of apyrexia, and accompanied by rheumatic pains, profuse sweats, marked anaemia and wasting, enlarged spleen, low mortality and a tedious convalescence.

#### Geographical Distribution:

This fever is now known to be much more widely spread than was at first supposed. It is by no means confined to Mediterranean townships.

Although much more common in Malta and Gibraltar than elsewhere, it is known to exist in countries bordering the Mediterranean, including Egypt and Tunis<sup>(25)</sup> and in the islands of the Mediterranean, in India, China, the United States, South Africa (Orange River Colony), Porto Rico (Musser & Sailer (Mr Cox)) and Manila (Strong and Musgrave). Some say that it never occurs primarily in this country, but Manson<sup>(1)</sup> and others have described cases which originated in England. The cases reported from the United States<sup>(2)</sup> were said to be imported ones.

Cases have also been reported from the Fiji Islands and South America.

In fact, generally speaking, it is diffused over a wide area within the sub-tropical regions.

#### Etiology:

When inquiring into this question we note that Malta Fever is not contagious, is essentially a disease of the summer months and is endemic in affected regions - occasionally epidemic.

#### Predisposing Causes:

No doubt anything which lowers the vitality increases the susceptibility to the fever: e.g. bodily fatigue, overcrowding, ill-ventilated or damp rooms,

overwork, lack of nourishing food and chills have all an important bearing on susceptibility.

It is now held by some <sup>(3)</sup> that people of a rheumatic diathesis are more prone to fall victims to the fever than others, and that, if they take it, the course of the illness, is, as a rule, severe and prolonged. There are some who have a special idiosyncrasy in relation to Malta Fever, and who do not take the infection, however much they are exposed to it.

#### Method of Propagation and Dissemmination:

Much attention has been devoted to this question during recent years, as everyone realises that once definite and satisfactory conclusions have been arrived at, preventative measures will soon follow.

Much has been done by the Malta Fever Commission <sup>(4)</sup> both experimentally and by observations on hospital cases.

It is now known that not only human beings, but also goats, mules, dogs, monkeys, mosquitoes and fleas may be infected by the specific micro-organism, but the effects of inoculation are seen most markedly in man.

The urine, faeces, milk, sweat, saliva and skin scrapings have all been examined for the *Micrococcus Melitensis*, and observers have come to the conclusion that the mode of exit from the body is chiefly

by the urine, and also in the milk and quite possibly also in the faeces, although it has not, as yet, been isolated in the latter.

The urine of patients suffering from Malta Fever is found to contain the micrococcus from the fifteenth day of the fever until convalescence is established.

Knowing the above facts, one is not surprised to learn that clothing, soil, dust and articles of food are contaminated with the micro organism. (Horrocks & Shaw in "Reports of Malta Fever Commission").

The next question is, how is the infection conveyed to the human body ?

The older authorities were of the opinion that the disease was directly due to the decomposition of animal and vegetable matters, in places where insanitary conditions prevailed, and that it was more particularly a faecal pollution. Opinion was divided as to whether infection was water or air-borne. Many thought the disease was got by breathing in foul air from drains. This may have a bearing on the causation, but only indirectly by lowering the vitality and so increasing the susceptibility to infection.

We, therefore, look to other possible modes of transmission, and the most important of these are:-

milk and other liquid and solid food, mosquitoes, dust, or by direct contact.

First consider dust as a factor in transmission:-

When we realise that not only man, but also goats, mules and dogs are subject to infection by micrococcus Melitensis, and that every infected case is excreting numerous cocci in its urine, we naturally think how likely it is that the dust which arises from the contaminated soil in dry weather should convey the infection. Experiments have been carried out in<sup>(5)</sup> which monkeys were confined in an atmosphere of dust heavily charged with the specific organism of Malta Fever. These animals which were subjected to a dusty atmosphere certainly developed the fever, but we must remember that the dust contained a much larger proportion of micrococci than is the case under ordinary circumstances. Besides, the infection might easily have been conveyed by direct inoculation, monkeys, as we know, having a great tendency to scratch themselves, and in that way entrance may have been obtained through some slight abrasion, or even through a crack in the mucous membrane of the nose or mouth.<sup>(8)</sup> There is, therefore, no direct proof that dust, in its natural state, conveys the infection.

Consider next transmission by Actual Contact:

Healthy subjects have slept in the same bed with infected ones and have also worn nightdresses which had been used by Malta Fever patients, and yet they remained free from the fever.

No doubt contamination with infected urine or faeces can produce an attack, provided there is some broken surface to admit the organism, but there is no evidence that contact of uncontaminated surfaces can lead to infection. (5)

Mosquitoes or Goats' milk must, therefore, be looked to as the possible agents in transmitting the disease.

As regards Goats' Milk:

From observations (9) made we may conclude that some goats in every herd in Malta suffer from Malta Fever and the micrococcus Melitensis is being constantly excreted in their milk and urine. Judging from experiments conducted by Horrocks and Kennedy (4) it is possible that the absorption of goat's milk by the alimentary tract may result in an attack of Malta Fever, but the incubation period, in the cases of fever which they thus produced, was a much longer one than occurs under ordinary circumstances - as much as two months in some cases.

There are certain facts in favour of goat's milk being the chief source of infection:-

- (1) Monkeys can be infected by feeding on goat's milk.(5)
- (2) An outbreak of Malta Fever occurred on board a certain ship (23) which had remained off Malta only long enough to take off a small cargo, which latter included a supply of goat's milk for the use of the ship.
- (3) Malta fever broke out in the Punjab in India and at the same time the presence of the micrococcus Melitensis was demonstrated in the goat's milk of the district.(6), (23) and (24).
- (4) Malta Fever in Rhodesia is said to have followed the introduction of goats there.
- (5) The occurrence of Malta Fever among children who consumed unboiled milk was found by Davis to be four times as great as that among children drinking boiled milk.

Notwithstanding these facts, there is no strong proof that goat's milk is the principal factor in the transmission of infection, and in opposition to the theory, Ross<sup>(7)</sup> states that four men fed on infected goats' milk did not develop the fever, and also that since the English forces at Malta took to boiling their milk the admission of Malta Fever<sup>cases</sup> to Hospital has increased instead of diminished, and the one regiment which did not boil its milk, had the lowest admission rate, strange to say.

That intestinal antiseptics have no power in diminishing the susceptibility to, or in cutting short the course of the fever is a fact which might be brought forward against the goat's milk theory.



With regard to goat's milk, it is now known that the micrococcus Melitensis may be excreted in the milk of an infected case for three months, without there being any symptoms of the disease in the goat or change in the appearance of the milk.

In an Editorial note on Malta Fever in the Journal of the R.A.M.C. 1906<sup>(20)</sup> it is stated that from experiments and observations made, there can be little doubt that Malta Fever is conveyed to man by means of goat's milk.

Now lastly let us enquire into the likelihood of Mosquitoes being the important agents in the causation.

J. Crawford Kennedy<sup>(10)</sup> is inclined to belittle the influence of Mosquitoes and says that in making enquiries at Valetta Hospital he found that nearly all the hospital attendants, in the ward for acute cases of Malta Fever, fell victims to the fever, but that for a number of years not one of the nursing sisters in this ward took the fever. He thinks that if mosquitoes had been the chief factor the sisters, who supervised the work of the ward, would have been as liable to infection as the attendants who attended personally on the patients. His explanation is that infection was conveyed by actual contact with the patients and their excretions.

Ross & Levick<sup>(11)</sup> although unsuccessful in their experiments with insects, are yet strongly in favour of the insect theory, and cite several cases where an attack definitely followed an insect bite, in people who had not previously been exposed to infection.

Although the micrococcus Melitensis is so seldom recovered from mosquitoes, examined for them, that is no proof against the mosquito theory, for one must bear in mind how scarce the organism is in the peripheral blood, and therefore how easily a mosquito may bite an infected person and yet not carry away the specific micro-organism. Were it otherwise, it is difficult to imagine how anyone in an infected district could escape.

E. H. Ross draws attention to the condition of affairs at Port Said. Here there are two hospitals. The English Hospital is situated in a low and filthy part of the town, but is well-exposed to the prevalent winds and has very little stagnant water in its immediate neighbourhood - both factors against the presence of mosquitoes. The Government Hospital, on the other hand, is very sheltered and has many pools of stagnant water where mosquitoes can breed.

On enquiring into the prevalence of Malta Fever at these two Hospitals, we find that the English Hospital is absolutely free from Malta Fever, while there have been many cases in the Government Hospital.

Horrocks and Kennedy<sup>(12)</sup> conducted experiments to find out the relationship of mosquitoes to Malta Fever. They came to the conclusion that the mosquitoes chiefly concerned were the *Culex Pipieus* and *S. fasciata*, and that *A. Zammitti* had probably no relationship to the *micrococcus Melitensis*. Only 1% of Mosquitoes yielded the organism, but that is not to be wondered at owing to the scarcity of the specific organism in the peripheral blood, as we have already pointed out. It was proved accidentally in the course of experiments that mosquitoes could convey the infection from monkey to man.

According to the incubation period, from experiments carried out by the Malta Fever Commission, the only class of experimental cases in which the incubation period was within the usual limits, was in that due to mosquito infection in which the average was eight to eleven days.

Lastly, the time of year when most cases of Malta Fever occur is in the hot dry weather and it is then that mosquitoes are also most prevalent.

Personally I am in favour of the mosquito theory and shall again refer to it when dealing with my own case.

Bacteriology:

The specific micro-organism - micrococcus Melitensis - was discovered by Bruce. It is a cocco-bacillus about  $0.33\mu$  in diameter, and usually occurs singly or in pairs, but when grown in broth it appears in short chains. Agar and gelatine cultures often consist almost entirely of bacilli. According to Durham some are somewhat curved, and according to Gordon flagellae may be present.

The micrococcus Melitensis is non-motile, stains easily with all basic aniline dyes, but rapidly loses its colour when treated with alcohol, and is not stained by Gram's method.

The bacteriological examination of tissue specimens is impossible at present owing to the absence of successful fixing agents. The micrococcus readily emulsifies, and is, therefore, well adapted for agglutinating experiments.

On a sloping surface of peptone agar -  $1\frac{1}{2}\%$  at a temperature of  $37^{\circ}\text{C}$ . its colonies become visible to the naked eye as minute transparent colourless drops on the surface. In about 36 hours they assume a transparent amber colour and in four or five days become opaque. No liquefaction occurs.

The micro-organism has been isolated from the spleen, blood, urine and milk.

Influence of Age and Sex:

Malta Fever may attack one at any age, but those most frequently affected are young healthy adults between the ages of six and thirty years. Children are much more rarely affected than young adults, and the aged less commonly still.

Women are more susceptible than men, in the proportion of seven to three.

Incubation period:

Authorities are, so far, not quite agreed on this question, but probably it is somewhere between five and thirty days according to the mode of infection - say 10 or 12 days on an average.

Horrocks quotes two days as a minimum and Col. Davies gives as much as six weeks for a maximum.

It is pretty safe to say that patients who remain free for, say two months, after leaving the infected area may be considered safe from attack.

Seasonal Prevalence:

It is an endemic disease never altogether absent, but now and then so prevalent as to constitute an epidemic.

If we refer to statistics bearing on the subject we find that Malta Fever is a disease of summer - most cases occurring during the months of May, June, July, August and September. - the maximum number being in July.

Dry summers are found to provide most cases of undulant fever.

Malta Fever does not occur to any extent during spring or until the rains have ceased, and it is found that a moderate but continued rainfall is better for prevention than an interrupted fall of similar amount.

The amount of rainfall is in inverse ratio to the number of Malta Fever cases. The virus which causes Malta Fever requires, besides dryness, a certain temperature. Although some cases do occur in tropical climes, a sub-tropical climate seems to be the most favourable.

The Sirocco (a south-east wind), or Levanter as I found it was called in Gibraltar, is common in Mediterranean towns, and its presence seems to be specially associated with the incidence of Malta Fever.

With reference to the Etiology of the fever in relation to seasonal prevalence, the fact that it is more common in the warm summer months rather points to mosquitoes as against goat's milk, being the cause, although if it is mosquitoes, we would expect even fewer cases to occur in the winter months than we find to be the case. We may also note that the seasonal prevalence of undulant or Malta Fever differs somewhat from that of malarial and enteric fevers.



The Question of Immunity from subsequent attacks:

Bruce has come to the conclusion that, as in many other infective diseases, one attack, does, as a rule, confer immunity.

Hughes<sup>(13)</sup> has not been able to find the records of a single case, of a subsequent attack of Malta Fever occurring in a patient who had already had one. He draws attention to the fact, that those short attacks of fever or febricula, lasting from seven to fourteen days, which are frequently met with in sub-tropical regions, do not confer immunity from more severe ones occurring later on. If a more severe attack should occur subsequently, we could not say definitely whether patient is suffering from a relapse or from a re-infection.

Hughes only considers a case one of true undulant fever, if the attack has lasted at least three weeks, or if a post-mortem examination has been held and confirmed the diagnosis. Many naval surgeons are inclined to think that one attack rather predisposes to another, and on this account those Malta Fever patients who are invalided from the Mediterranean, are not sent back until several years have elapsed.

No doubt many of the so-called fresh attacks of short duration are just relapses.

The fact that very few of the adults of the native population of Malta suffer from attacks of the fever is ascribed by some to the fact that they have suffered from it more or less severely in youth or childhood. We have no reliable data on which to say definitely when a patient becomes proof against relapses.

To sum up, I think we may say that one attack does confer immunity, at least for a considerable number of years.

#### Clinical types of Malta Fever:

Three types are commonly described, viz:- the Malignant, Undulatory, and Ambulant or Intermittent.

Birt and Lamb<sup>(14)</sup> sub-divide the malignant type into two other types - one in which the case is a severe one from the onset and causes death in a week or so, and another which is less acute and more prolonged and usually proves fatal after one or two relapses have occurred.

#### Symptoms:

##### I. General.

##### (a) In Malignant Type.

The onset is usually sudden and severe. Patient complains of severe headache, nausea and perhaps vomiting. He is "sore all over", has a



very feverish look and his tongue is thickly furred on the dorsum, the edges and tip being bright red. Patient's breath is very offensive, he has tenderness in the epigastric region and also in the hepatic and splenic regions sometimes. Bowels may be constipated or loose, and in the latter case the prognosis is very grave. The stools are offensive. The temperature is found to be in the region of  $104^{\circ}\text{F.}$ , but may come down almost to normal for a short time, only to run away up again perhaps as high as  $110^{\circ}\text{F.}$  before death.

Hughes<sup>(13)</sup> states that the temperature may abate after the first few days and remain about  $102^{\circ}\text{F.}$  but a condition of extreme nervous excitability sets in at the same time. This may lead to a condition of extreme nervous exhaustion - the higher nerve centres being evidently poisoned - and death may result.

In the cases which prove fatal at an early period, say, about the 10th day, death is usually due to hyperpyrexia, whereas in those which Birt and Lamb classify as type B., where death occurs after one or two relapses, the fatal termination is usually a result of pericarditis or pleurisy with effusion, heart failure or the supervention of some such disease as phthisis.

### B. The Undulatory Type

This is the most common form in which Malta Fever occurs. Its onset is usually somewhat indefinite, the symptoms being rather obscure to begin with. The patient feels out of sorts and low-spirited and complains of nausea, loss of appetite, headache and pains in the back - particularly in the cervical and lumbar regions. It is very unusual for the fever to be ushered in by a rigor or vomiting. The patient gradually becomes more and more miserable and has creepy and chilly sensations, but fights against them for some days, attributing his troubles as a rule to his stomach or bowels being out of order, for he notices that his tongue has become thickly furred, enlarged and indented laterally by his teeth, that he has a bad taste in his mouth, an uncomfortable sensation of tenderness in the epigastric region, and his conjuncturae may be slightly jaundiced. The bowels are usually constipated, but sometimes they are loose and offensive and even streaked with blood and mucus.

Realising that he is becoming steadily worse, he at last gives in and takes to bed and on his temperature being taken we usually find the thermometer registers somewhere about 103° or 104°F.

If the temperature curve in this undulatory type is studied, we notice that it is very irregular, consisting, as Hughes says, of intermittent waves or undulations of pyrexia, of a distinctly remittent character. Also, that the course of the fever is very irregular, and as a rule very prolonged, not unfrequently lasting for many months. The individual waves of pyrexia vary much in length sometimes lasting only a few days, at other times continuing for three or four weeks.

Two of the outstanding features of this type of fever are the very irregular and erratic nature of the temperature curve, and the low mortality notwithstanding the prolonged and apparently very serious illness.

If we examine the patient when he has at last taken to bed, we observe that his attitude is one of extreme listlessness and depression when the temperature is inclined to be down, but when it rises, this condition is replaced, very often, by one of restlessness, the patient appearing anxious, ill at ease, and often much troubled with sleeplessness.

In the earlier stages there is often some pharyngitis, nasal catarrh and slight cough.

As the disease progresses the patient becomes pale and wasted, and worn out with the profuse

perspirations which follow the rises of temperature, but he is fairly comfortable and begins to sleep better. When nausea is absent, the appetite is wonderfully good and plenty nourishment can be taken, although it does not seem to do the patient much good.

In some cases after a few weeks have elapsed, the temperature gradually settles, steadily becoming nearer normal in the morning, then rising less in the evening until it reaches normal both morning and evening, except for an occasional rise at night, every few days. A period of subnormal temperature may follow and patient may then pass into the convalescent stage. On the other hand, the temperature curve may just touch the normal and then gradually rise again, or begin to rise without ever having come quite down; or, after a period of normal temperature, lasting a few days as a rule, a relapse, which is so typical of this fever, may occur, and <sup>the</sup> patient may pass through a period of pyrexia as before. Any number of relapses may occur.

In more severe cases, what troubles the patient just as much as, or even more than the feverish state, are the local symptoms. These are often very painful and persistent, consisting usually of severe neuritis, arthritis and orchitis, which

shift about, take their own course, and are but little affected by any treatment which may be adopted.

While suffering from these symptoms the patient may experience intervals of more or less complete relief from them, but any increase of symptoms is usually associated with a corresponding increase of temperature. When the temperature has subsided altogether, the chief symptoms are a result of anaemia and debility.

C. The Intermittent or Ambulatory type:

In this variety the patient may never be so ill as to be incapacitated from following his employment, and no unpleasant symptoms may occur, although there is usually considerable malaise and headache, etc.

The temperature is the chief indication that the patient is not in his usual state of health. There is a daily pyrexia, the rise occurring in the afternoon or evening, a normal temperature being again recorded in the morning. In this type, once the temperature has settled down again, and remained rather subnormal for a few days, there is much less chance of relapse than in the other types. But as long as the fever is present, it is liable to assume a more severe form, especially if

the patient is indiscreet in his diet, or careless as to his general health.

#### Principal Symptoms in Detail:

##### Facies:

At the commencement of the fever, or if painful complications are present, the patient exhibits a pained, anxious and worried look - quite an agonised look sometimes, if there is severe neuritis or arthritis.

The expression later on becomes listless and apathetic, indicating the sufferer's despondent frame of mind. In some very prolonged cases the patients self-control gives way and he becomes tremulous, peevish and easily moved to tears.

The complexion is pale, sallow and even yellowish.

##### Tegumentary System:

The skin after the initial congestion becomes pale, sallow and even jaundiced in some cases. No typical rash accompanies this fever although sometimes a few pimples come out on the abdomen, and sudamina is very common. Patients often suffer great discomfort from the prickly heat (which is so general in the hot season in sub-tropical climates,) but not to any greater extent than healthy people do.



Sub-cutaneous petechial haemorrhages of a purpuric character have been described by several observers. These are probably largely due to the marked and long-continued anaemia, which is invariably present.

The hair usually falls out in crops, in the later stages and patient may become bald in patches and grey hairs frequently come in. On recovery the hair often grows more abundantly and stronger than ever.

Not only is there a casting off of the hair, but there is also a desquamation of the skin in most cases.

In some this is very marked, in others only slightly - perhaps limited to the palms of the hands and feet. The nails become dry and brittle, and a nail may be markedly grooved transversely, the grooves corresponding to the periods of pyrexia.

Profuse perspiration is a very marked symptom as a rule. The skin is in its natural state in the forenoon, but becomes dry and burning-hot as the temperature rises in the afternoon or evening, and then bathed in perspiration when the exacerbation passes off. The sweat simply runs off the patient, soaking the pillow, bed-clothes and night-dress, the latter having to be changed four or five times within a couple of hours in some cases.

Apart from the daily diaphoresis associated with the exacerbations of temperature, profuse perspirations may originate as a result of fatigue or emotion, more especially in nervous and highly-strung individuals. The sweating may not be general all over the body, but may be limited to one particular region such as the legs.

When dealing with the skin we may mention that an unpleasant and peculiar mousey odour is sometimes emanated from it. It is not in any way associated with dirt or uncleanness, and somewhat resembles the odour which is present in some cases of typhus. It tends to disappear in the later stages of the fever, but is a source of much annoyance to the patient when present.

#### Digestive System:

The entire alimentary tract is more or less affected, being in a state of congestion and catarrhal inflammation. The pharynx and tonsils are inflamed and swollen, giving rise to much pain and discomfort on swallowing, and sometimes a genuine attack of acute tonsillitis occurs in the initial stage of the fever. The gums may get into a very unhealthy and spongy condition and bleed on the slightest provocation.



The tongue is swollen, flabby and indented, covered with a thick fur at first, but later clearing up at the edges and tip to leave them red. When there is a high temperature, with much constitutional disturbance, the tongue becomes dry and brown and sometimes very foul.

The tongue is a reliable guide to the general condition of the patient and does not become thoroughly clean until convalescence is established.

The congested state of the digestive tract is associated with nausea, loss of appetite, bad taste in the mouth, pain and discomfort after food, together with epigastric tenderness. Sometimes the intolerance of food is so great that the ingestion of any is followed by vomiting of a very persistent type.

Hughes<sup>(13)</sup> says that the tenderness is most marked in the epigastric notch and towards the left hypochondrium, but in my small experience it has been more towards the right side.

Associated with this tenderness there is often considerable enlargement of the liver and spleen, both of which may be palpated below the costal margin, especially in the early months.

The enlarged liver is in a state of congestion with cloudy swelling of the liver cells and there is an infiltration of small round cells in the interlobular fissures.

The bowels are usually constipated and in prolonged cases may become markedly so, the passage of hard Scybalous masses being attended with great pain. The stools, if inclined to be loose, are bulky, greasy and extremely offensive.

Diarrhoea may occur, and if severe, is a grave symptom. The faeces are usually dark in colour, and very seldom present the pea-soup ~~chain~~ features of the typhoid stool.

Strings of mucus may be passed, especially when symptoms have abated and patient perhaps has been allowed a too generous diet; at times the stools are streaked with blood. There may be considerable tenderness over the intestines, but tumidity, tympanites and gurgling are extremely rare symptoms. As recovery takes place digestion improves and in mild cases is wonderfully good throughout the fever. There is none of the ravenous hunger which occurs in the convalescent stage of typhoid.

#### Nervous System:

The toxins formed by the micrococcus Melitensis seem to have a special affinity to nerve matter, and produce temporary aberrations, which, in some cases, closely simulate permanent lesions of the central nervous system or spinal cord. At the commencement of the illness, delirium and sleeplessness occur in

about 15% of the cases, and severe headache, especially of the frontal type, is common.

There is often marked irritability of temper, and some loss of memory. For some time after the severer attacks - according to Lane Notter - memory is considerably impaired, especially with regard to names and dates - or rather the chronological order of events.

Anaesthesia, hyperaesthesia, parasthesia, paresis or even paralysis may occur, but are of a temporary nature, though sometimes persisting for months. Pain is often a marked symptom and is due to neuritis and arthritis in most cases. When due to neuritis it is most commonly met with in the lumbar region, over the sacro-iliac joint, at the point of emergence of the sciatic nerve on to the buttock, and within the pelvis the nerves supplying the rectum and pelvic floor are most frequently affected.

Noises in the head are sometimes very persistent and the cause of much discomfort.

As the fever progresses the patient, in the severer cases, becomes weak, tremulous, peevish and easily moved to tears, requiring very gentle and patient nursing. The nervous system in severe cases is more or less wrecked for the time being, but with careful treatment in the convalescent stage, recovery

is rapid and wonderfully complete.

Circulatory system:

The heart is rarely directly affected, but its nervous mechanism is. The pulse is little above the normal rate to begin with (from 80 to 90 per minute, as a rule) but it gradually increases in frequency and loses in impulse, until about the sixth week it is often between 110 and 120 per minute.

Much depends on the patient's nervous temperament, those who are highly strung very frequently suffering from palpitation.

The highest pulse rate in a non-fatal case which Bruce has observed was 132 per minute, on the 78th day.

Some observers<sup>(15)</sup> have drawn attention to organic changes of an inflammatory nature affecting the heart in the course of the fever, both endocarditis and pericarditis having been noted, but the number of such cases is comparatively small.

Haemic murmurs may be audible.

Respiratory system:

Epistaxis occurs in less than a fourth of the cases, and arises from the congestion which is present throughout the respiratory apparatus.

Cough often persists to a greater or less extent throughout the illness, and is sometimes very severe

and accompanied by a profuse expectoration which may be streaked with blood.

The symptoms are often out of all proportion to the physical signs.

On auscultation the breath sounds may be practically normal, but in some cases they are harsh and accompanied by rhonchi and sibilant râles. These evidences of congestion are often most marked at the apices. On percussion - areas of diminished resonance may be made out, but a condition of pneumonia only occurs in about 2% of the cases, and in these pleurisy with effusion may supervene.

In the severer cases of respiratory involvement where the vitality has been much lowered, the inflammation may pass into a condition of chronic tuberculosis.

#### Genito-urinary system:

The kidneys are congested and in a state of glomerular nephritis. The urine is at first dark in colour and often loaded with lithates and phosphates, and is diminished in amount, but only in the very severe cases does it contain albumin.

When there is great constitutional disturbance, with marked congestion of the liver and other abdominal organs, we may find bile in the urine.



Later, the urine exceeds the usual quantity, and is pale, clear and of a low specific gravity.

Neuralgia or inflammation of the testicles are very common complications of Malta Fever, orchitis being an extremely painful affection. The testicle and epididymis swell up rapidly to the size of a small orange, there is often some effusion into the tunica vaginalis and the skin of the scrotum becomes red and inflamed. The inflammation undergoes spontaneous resolution in the course of a few days, as a rule, but may recur if patient goes about too much before convalescence is thoroughly established.

#### Haemopoietic system:

As we have already stated profound anaemia is often present in Malta Fever, and on examining the blood, we find a marked diminution in the number of red blood corpuscles: anything from three to three and a half millions being common. The leucocytes are not much affected, although in some cases there is an increase to about eleven or twelve thousand.

The spleen weighs about 18 oz. on an average, and is soft and diffluent. The Malpighian bodies are enlarged from apparent increase in the number of round lymphoid cells. A condition of intense congestion is present.

The spleen itself is always enlarged and may be tender on palpation, and sometimes severe pain in the

splenic region is complained of.

Arthritic system;

In the early stages of the fever, and more often during the decline than the rise of a pyrexial wave, one or more of the joints may be affected by an acute non-suppurative synovitis. This occurs in about 50% of the cases, is usually of sudden onset, and is attended with much pain and swelling, but no redness as a rule. The condition never goes on to suppuration, although the effusion may be considerable. The shoulders and knees are most frequently affected and next the hips, elbows and wrists. When the vertebral or sacro-iliac joints are affected the pain is excruciating. The condition may simulate tuberculous sacro-iliac disease. The patient is often terrified to move and may neglect to move his bowels for days, in order to avoid the movement which the act would involve. Pressing the ilia towards the middle line, or pressure over the sacro-iliac joints, cause severe pain.

Joint affections of a less severe type are commonly met with in the later stages of the fever, when the pyrexia is less marked, and are of the nature of a subacute synovitis. The pain is proportionate to the tension. The bursae are sometimes affected, especially the bursa over the patella becoming filled with fluid and very tender.

Some writers describe painful node-like swellings occurring on the ribs or their cartilages and on the sternum, with no history of syphilis to account for them.

As long as there is any rise of temperature, so long is the patient liable to an exacerbation of joint symptoms.

Though some permanent stiffness may remain, recovery is the rule.

#### Special senses:

Hearing may be impaired, often one ear alone being affected, the deafness being accompanied by noises in the head sometimes. Vision is not often impaired, but if it is, it rapidly improves with the establishment of convalescence. Taste and smell are not often affected, though in some cases the patient experiences smells and tastes which are not actually there.

#### Pyrexia:

We may make one or two remarks on the pyrexial condition, although we have already dealt with it, when discussing the various types of fever.

The fever is practically never ushered in with rigors or paroxysms of any kind, but the patient can usually tell that a rise of temperature is going to take place on account of his usually experiencing



a sense of chilliness, with coldness of the extremities, and an uncomfortable creepy or crawling sensation up the back of the spine, at the same time feeling very miserable and out of sorts.

In the most typical cases the temperature curve resembles that of enteric fever, in its steady rise and fall.

Although pyrexial undulations are more or less characteristic, they are not by any means always present, and every variation in the length and character of the waves and daily temperature curves may be met with.

In mild cases the temperature may be intermittent - coming down to or almost to the normal every morning, and in more severe cases regular waves of pyrexia occur which may or may not be remittent in character. The fall may be a gradual one by lysis or an abrupt one by crisis.

Most frequently the rise and fall of the pyrexial waves is gradual and associated with more or less remittance or intermittence.

The length of the individual waves varies very much. There may be several waves in one attack or the initial wave may persist throughout.

Most cases end with a shorter or longer period of intermittance, which in turn may be followed by a period when there is only an occasional rise of

temperature in the evening, but as long as the patient is subject to these slight rises, he may suffer from a relapse of any severity. Hughes says that one of the surest signs of permanent recovery is a period of subnormal temperature lasting from one to six or more days and associated with a clean tongue. With this observation I very thoroughly agree.

A continuously high temperature curve is common in the malignant and fatal types and in these cases hyperpyrexia - as high as  $110^{\circ}$  or  $112^{\circ}\text{F.}$  may be registered shortly before death.

Frequently in the secondary stages of the fever, the temperature is of the remittent type during the pyrexial periods. In the early morning and forenoon the temperature is very often between  $98^{\circ}$  and  $101^{\circ}\text{F.}$ , but in the afternoon it rises from two to four degrees, and continues high until midnight or a little later when a rapid fall takes place, accompanied by profuse sweating.

#### Complications:

Several complications have been dealt with under the different systems, but there are one or two not commonly met with which have yet to be described.

When discussing the affections of the digestive tract we mentioned that the stools were sometimes streaked with blood. Although that has been admitted

to occur somewhat frequently, yet, until recently, no severe haemorrhage with ulceration of the intestine was believed by Hughes ever to occur.

In his monograph on Malta Fever<sup>(13)</sup> he states that one of the differences from typhoid fever is the absence of ulceration of Peyer's patches. He describes patches of congestion occurring throughout the small intestine, but states that Peyer's patches are unaffected, except by the general congestion, and in no case has he found ulceration present.

Lieut. Bousfield in the "Lancet" of 1906 states that he has had several cases under his care in which there was more or less profuse haemorrhage from the intestine. One patient passed about a pint of blood in 36 hours. He draws attention to the fact that the mesenteric glands are often enlarged in Malta Fever and the micrococcus Melitensis found in them, and he does not see any reason why they should not cause ulceration of the neighbouring intestine, as occurs in typhoid. He cites a case in which typhoid fever was suspected to be running concurrently with Malta Fever, but the blood only reacted to Malta Fever, and the spleen and mesenteric glands yielded pure cultures of the micrococcus Melitensis, but the bacillus Typhos. was not obtained. The abdominal viscera were in a state of acute congestion, and in the small intestine there were eight ulcers

within 36 inches of the ileo-caecal valve, some of which corresponded to Peyer's patches. There was also a sub-mucous haemorrhage in the sigmoid flexure of the large intestine.

A case of intestinal haemorrhage occurring in a case of Malta Fever was also reported by Staff Surg. Reid in the British Medical Journal 1906. In it the haemorrhages were copious and repeated, but the patient eventually recovered.

Since writing his monograph Hughes has found ulcers in three out of sixty-two cases where a post mortem was held. The ulcers were situated in the ileum, colon and rectum.

Some of the ulcers corresponded to the situation of Peyer's patches.

Purpura haemorrhagica was described by G. E. Macleod<sup>(16)</sup> as occurring in the course of Malta Fever. Since then several other cases have been met with; no doubt it is associated with the profound anaemia which is developed.

Differential Diagnosis:

Malta fever has to be diagnosed from typhoid fever, malarial fever, tuberculosis, the hectic fever of suppuration and from a class of fever called simple ardent fever or febricula which is very common in sub-tropical climates. Probably many cases of fever of the undulant type lasting less than three weeks are abortive or mild forms of enteric or malta fever. Some cases may go on for five or six weeks before the agglutination test yields positive results, especially if it follows some other malady such as influenza, therefore persevere with your tests for some weeks in spite of negative results.

(1) Malta fever from Typhoid fever:

Malta fever is of longer duration: there is, as a rule, constipation instead of diarrhoea, and it is only in exceptional cases that the stools resemble those of typhoid fever. There is no tumidity of abdomen, no gurgling in iliac fossae and no rose coloured spots in typical cases. There are painful articular, neuritic and orchitic complications, and often a peculiar odour about a Malta fever patient, and the mortality is much lower than in enteric fever. In typical cases the temperature curve has a steadier rise and fall in typhoid, but some cases, especially of the severe type, cannot be diagnosed from Malta Fever, in the early weeks,



unless a "post-mortem" is held. The number of such cases is comparatively small and confined to the severe and fatal cases as a rule.

Cases of both diseases running concurrently may occur, and be even attributed to a similar faecal origin.

Hughes states in his book that the total absence of morbid changes in Peyer's patches distinguish Malta fever from enteric, where such lesions are so common, but we now know that ulceration of intestine may occur in Malta Fever.

There are other means of diagnosing Malta fever from enteric.

If we examine the splenic pulp recovered from fatal cases or removed by syringe from living cases, we may find the micrococcus Melitensis, or the Bacillus of Eberth & Goffky which latter is invariably absent from the spleens of Malta Fever cases, and is supposed by many to be the proximal cause of enteric fever.

The serum reactions of these two fevers yield (26) valuable and reliable results. The results, with respect to their causative organisms, are such as could only exist in two distinct forms of fever. The seasonal prevalence of Malta Fever and enteric fever is also not identical. When the two diseases run concurrently, it is very easy to overlook one

of them, and the blood may fail to give a positive reaction to one of them. J. C. Kennedy cites a case<sup>(17)</sup> in which the blood reacted to typhoid fever but not to Malta Fever (in a dilution of 1 in 10), and yet six days after, an abundant growth of micrococcus Melitensis was obtained from the spleen. In that case the micrococcus Melitensis was found to be more abundant than the Bacillus Typhos.

It was doubted, at one time, whether the two diseases could occur together, but it is now an established fact that they can. J. C. Kennedy has drawn attention to two cases<sup>(18)</sup> which illustrate this. In one, the patient passed through a typical attack of enteric fever, the blood serum reacting completely to enteric fever in a dilution of 1 in 50 and not at all to Malta Fever in a dilution of 1 in 10. After the temperature fell to normal it rose again and the serum gave immediate and complete reaction to Malta Fever and incomplete to enteric in a dilution of 1 in 10.

In the other case the blood reacted first to Malta Fever, and later failed to react to Malta Fever, but a slight reaction to enteric was present on the latter occasion.

It is, therefore, readily understood that the serum diagnosis is not absolutely to be relied on, as the presence of one fever may mask that of the



other, should they occur together, and it is, therefore, very important that all cases of Malta Fever should be very carefully treated, in case enteric should also be present, although giving rise to no typical symptoms.

It seems that the agglutinins of the one fever tend to destroy those of the other, and Dr Phillips of Cairo quotes a case <sup>(19)</sup> in which the serum, when first tested, agglutinated the Bacillus Typhos. and micrococcus Melitensis, and a week later it still agglutinated the micrococcus Melitensis, but failed to react to enteric.

Of course it is well known, that the fact that a patient has suffered from an attack of enteric fever does not in any way protect him from a subsequent attack of undulant fever and vice versa.

In suspected cases of Malta and Typhoid fever, we may learn something by examining the excretions, more especially the milk and urine, as these are known to contain the specific micro-organisms.

## (2) Malta Fever from malarial fever:

The difficulty of distinguishing these two fevers from one another is not nearly so difficult as in the case of Malta and enteric fever, although there is a resemblance in the remittance and intermittence of the temperature curve in both.

A careful study of the case will usually clear

up any doubts as to which is actually present. The fever, in the case of Malta fever is unaccompanied by rigors or paroxysms, the temperature curve although irregular in both, is of a more periodic and paroxysmal nature in malaria: the usual complications of Malta Fever are absent in malaria, and the examination of the blood usually reveals the malarial parasite in the latter.

Once the patient has completely recovered from Malta fever, he is not subject to the recurrences which are so common, even years afterwards, in malaria.

The seasonal prevalence of the two fevers is not identical, and again, the administration of quinine has a marked effect in reducing the temperature in malaria, but is powerless to influence the course of Malta fever.

(3) Malta fever from Tuberculosis and the Hectic fever of suppuration and Hepatic abscess.

These have only to be mentioned to be dismissed. It is possible that one might fail to make a correct diagnosis between them on a hasty examination, but careful observation and a thorough examination of the patient together with the application of the agglutination test to his blood should soon remove any doubts.

There is, of course, enlargement of the

liver in hepatic abscess, and should there be any possibility of its presence, an exploratory puncture should be made at an early stage, as it is then that an abscess can be most successfully treated.

(4) From cases of undulant fever met with in this country. In addition to the above diseases which have

been mentioned as more or less closely resembling Malta Fever, there is an undulant type of fever sometimes met with in this country, which has many points in common with Malta Fever. Such cases may only be differentiated by applying the agglutination test, and examining the blood and excretions for the specific organism - micrococcus Melitensis. Such cases afford great scope for careful study, as little or nothing is known of their etiology so far. They are important cases in so far that they cause much distress, have a very prolonged course and quite incapacitate the patient from following his employment. All the organs having been found, by careful examination, to be sound, and no apparent cause being detected, it is customary to say that the patient is probably suffering from a toxæmia, the result of gastro-intestinal sepsis, the dregs of influenza, etc. etc. Quinine, antipyrin, phenacetin, salicin, santonin and various intestinal antiseptics have been prescribed for such cases, but as a rule, they are little, if at all, affected by drugs.

The following two cases illustrate the type of case which I refer to:

Case I: G. H. age 31. Labourer.

Had been ill for nine weeks previous to date of admission to the Royal Infirmary, and when admitted complained of sickness, nausea, pains in the head and stomach, constipation, sweatings and occasional rigors and fever in the evening.

On examination:

Marked emaciation and anaemia.

Spleen and liver much enlarged.

Epigastric tenderness.

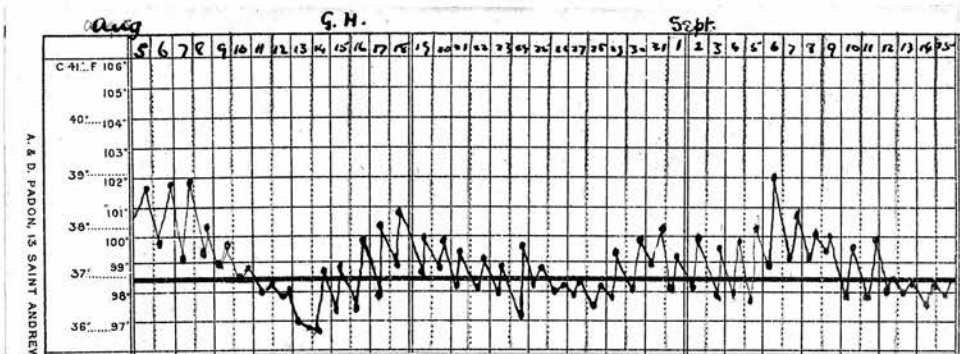
Blood: R. B. C. = 3,700,000 per c.m.  
W. B. C. = 10,000

Heart: healthy.

Lungs: Slight moist crepitations and  
rhonchi towards bases.

Patient had never been abroad, but had been engaged at work in drains, previous to his illness.

His blood was negative both to the typhoid and Malta Fever agglutination test. He was kept on milk and soda water at first, and later, on simple milk diet.

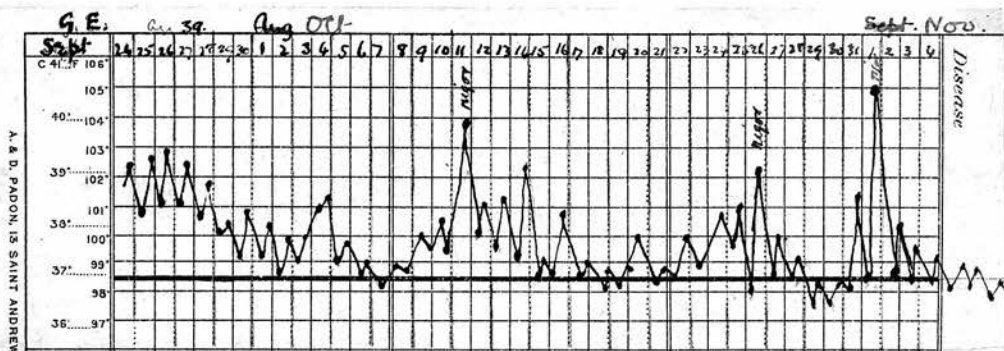


Anitperiodics and intestinal antiseptics such as Quinine and Liq. Hydrarg. Perchlor. and Salol were prescribed, but these had no apparent effect.

His chief symptoms were nausea, gastric discomfort and daily pyrexia followed by profuse perspiration. He was suffering from a wave of pyrexia when admitted and that was subsequently followed by two more waves. The temperature at these times was remittent and intermittent. He was strictly confined to bed, and at the end of seven weeks he was quite free of fever and had no subsequent relapses.

Such a case, lasting as it did altogether for about sixteen weeks resembles very closely a rather mild case of the undulatory type of Malta Fever. The nature of the temperature curve, the nausea and epigastric tenderness, the profuse perspirations and constipation and the enlarged liver and spleen were all points of resemblance; but the negative result of the serum test, the absence of joint or other painful complications, and the occasional occurrence of rigors were against the likelihood of the case being one of Malta Fever. The fact that the man had been employed at work inside drains is strongly in favour of the poison, whatever it was, having been obtained from that source.





Case II:

The second case was that of a man - G.E. - a tailor: age 39, who took ill on the 20th September, took to bed on the 22nd, suffering from headache, shivering, abdominal pain and discomfort in epigastrium, with sickness, nausea and slight bleeding at the nose.

His case was diagnosed as enteric fever and he was treated accordingly, receiving daily irrigations of the lower bowel: confined to a milk and water diet with occasional doses of castor oil.

On examination: (Patient is a large and heavy man).

Abdomen is distended.

Tenderness in epigastrium.

Furred tongue.

Spleen and liver enlarged.

Pulse quick and dicrotic.

Respirations - normal.

Reaction to Widal test was negative.

There was nothing in patient's condition to account for the pyrexia. The temperature was of the remittent type to begin with, never falling below 100°F. for the first few days, and the daily rise was followed by profuse perspirations.

Accompanying the second wave of pyrexia - which followed very closely on the first - were vague neuralgic pains in the back and limbs and

pains and swelling in the left knee-joint. At this time patient had a moderately severe rigor, and later on had two more rigors. After six weeks illness the patient suddenly took a turn for the better and had no more recurrences of the fever.

We must note that this patient's blood was not tested for the Malta Fever reaction, and we, therefore, cannot be certain that he did not suffer from that fever, although it is very unlikely that he did, considering the fact that he had never been abroad.

The temperature curve is not perhaps very typical of Malta Fever, the apyrexial intervals being very short, the temperature in the second and third waves too rapidly falling from a very high point to normal, and rigors occurring.

Still it illustrates my point, which is, that there is a class of undulant fever in this country which closely resembles Malta fever, is of indefinite duration, accompanied by much gastro-intestinal disturbance, unaffected by treatment and with a low mortality. Neuralgia and arthritis - such frequent complications in Malta Fever of the undulatory type - may or may not be present.

The result of the serum reaction whether negative or positive to the micrococcus Melitensis, will not influence treatment much, which, in such

cases must always be expectant; but it will assist us in arriving at our prognosis, which, from the patient's point of view is something gained.

Prognosis:

Although this fever may pursue a very prolonged and tedious course, and is capable of causing very distressing and painful symptoms, reducing the patient to a condition of extreme debility, yet, as far as life and ultimate restoration to good health is concerned, the prognosis is good.

The mortality is stated to be about 2% amongst soldiers, and 8% or 9% amongst civilians<sup>(20)</sup> - some state 13% in army at Malta.<sup>(22)</sup>

The average duration of the fever is stated to be from 70 to 90 days, which means that it may last from a month or two up to a year or two.

When called into a case of Malta Fever, we can never give any very definite opinion as to how long it is likely to last, but should the patient's temperature remain subnormal for five or six days and his tongue be quite clean, we have every reason to believe that he is on the point of recovery.

The largest number of deaths occur in the first two months, and are most commonly due to hyperpyrexia, heart failure or pulmonary congestion.

A case which is severe from the outset does not last long as a rule, and a gradual fall of temperature is much more favourable than a sudden drop.

The tongue is a valuable guide to the patient's condition. A dry brown tongue at any period is a bad sign, and as long as it is of an angry red colour at the tip and furred on the dorsum the patient is still suffering from the infection; but a clean moist tongue augurs well. The complications such as orchitis, neuritis and arthritis do not persist very long as a rule, and although the patient may be subject to pains and stiffness for some time after convalescence is established, they ultimately disappear completely in most cases.

Convalescence, once it is fully established, is usually rapid.

One attack does not predispose to another, and some authorities consider that a second attack never occurs<sup>(21)</sup> - many so called second attacks being simply relapses.<sup>(13)</sup>

Bruce is of the opinion that once a patient has recovered completely, he will never suffer from a recurrence of the fever - in other words,

that one attack confers immunity from subsequent attacks.

Some naval surgeons, on the other hand, are of opinion that one attack rather predisposes to a second and for this reason those invalided from the Mediterranean are not sent back until many years have elapsed. The fact that the adult population of native Maltese is comparatively free from Malta fever is ascribed by some to the fact that slight attacks in childhood or youth have conferred subsequent immunity.

It is probable that one attack does confer immunity, at least for a number of years.

It is impossible to say how long a time must elapse before a patient can be said to be proof against relapses.

There is no evidence that the fever has a deleterious effect on the health in after years or that it shortens life.

Birt & Lamb<sup>(14)</sup> point out that the agglutination reaction may be relied upon to aid us in our prognosis. They state that prognosis is unfavourable in cases which are severe from the outset with a low agglutinative reaction, or in cases where the agglutinative reaction rapidly falls from a high to a low figure, somewhere about zero.



A persistently high and rising agglutinative reaction would be very favourable, and augur a speedy recovery without relapses.

In these cases where the course is very tedious and attended with complications, the high reaction dwindles down gradually.

The opsonic index and the phagocytic index may also give us a certain amount of information. Both are definitely lowered in Malta fever.

#### Pathological anatomy:

This has been dealt with under the various systems.

#### Serum Reactions:

Professor A. E. Wright established the fact that the serum of patients who are suffering from, or who have recovered from Malta fever, produces a specific agglutinating reaction on the micrococcus *Melitensis*. He has further shown that dead cultures of the bacteria can be employed with the same result as living cultures. (27)

The specific agglomerative power of the blood persists, in the case of Malta fever, for

many years. Birt & Lamb have drawn attention to a case where complete reaction in 20-fold dilution was obtained seven and a half years after the attack.

The amount of agglutinin tends to be markedly increased during the periods of comparative apyrexia, and almost disappear from the blood shortly before death.

The highest dilution to give the agglutination is about a 2000-fold dilution and occurs in those attacks which are sharp, but do not last long, and which are unaccompanied by complications and followed<sup>by</sup> only slight, if any, relapses.

Those cases which are severe and end fatally only react in a very low dilution, the highest being 1-100.

In the long tedious cases the agglutination reaction is high to begin with, but gets lower and lower as time goes on, until it becomes very low indeed, and if the test is made for the first time, only towards the end of the illness, the reaction may be completely absent, but in that case does not negative a diagnosis of Malta fever.

The milk of infected goats has been found to cause agglutination of the micrococcus Melitensis, and this reaction is said to be a surer test of cocci being excreted in the milk than the serum reaction of the same goat.

TREATMENT:(I) Prophylactic:

Render one's surroundings as sanitary as possible. Keep goats well away from human habitations, as it is more than probable that mosquitoes can transfer the infection from goats to man. Prevent goats parading the street, as they no doubt pick up rubbish infected with urine, etc. Destroy all infected goats, i.e. those goats which give the milk or serum agglutination test. It is not enough to test the milk as the presence of the micrococcus in the milk is intermittent, and altogether absent during the first two or three months from the date of infection, during which time there may be no symptoms of disease in the goat and no change in the appearance of the milk.

If the fresh milk of goats or cows is used in areas where Malta fever is endemic, it should be subjected to a heat of 68°C. for ten minutes in order to destroy the micrococcus Melitensis. People are now using much more condensed milk than formerly. As the fever is evidently closely associated with the presence of mosquitoes, those should be destroyed as completely as possible. The best method to destroy the larvae is to pour 15 c.c. (per sq. metre) of petroleum on the surface

of all stagnant water, and to repeat this every 15 days in hot weather.

Malta fever should be treated as an infectious disease, and all cases should be isolated. In every hospital where Malta fever patients are admitted, the beds should be provided with mosquito curtains, and, as an extra precaution, all clothing, bedding, and excreta should be disinfected.

Europeans who have not been long in residence in infected areas should spend the hot and unhealthy season away from the Mediterranean if possible, and reside in more temperate climes. If this is impossible they should try and get to the country districts outside the towns.

See that the water supply and drains are in good order, and lead as hygienic an existence as possible, avoiding fatigue, chills and worry is advice worth following.

The systematic inoculation of Malta Fever antitoxin, in infected areas, will no doubt at some future date be employed as a prophylactic and be found to be thoroughly efficacious.

#### (II.) Therapeutic and General:

All the likely drugs in the Pharmacopoea have been tried at one time or another, but no specific remedy has been discovered so far. In fact such

drugs as Quinine and the Salicylates seem to have, if any thing, a rather deleterious effect.

We must just do our best to alleviate the different symptoms as they arise. The more commonly used drugs are Quinine, Salicylic acid, salicylates of soda, and quinine, antipyrin, phenacetin, phenalgin, salol and thymol.

Quinine irritates the stomach, upsets the digestion and frequently gives rise to unpleasant head symptoms and exerts no influence on the temperature.

The Salicylates are gastric irritants and cardiac depressants, and do not succeed in alleviating the painful complications.

Antipyrin has the power of reducing the temperature, but the effect is only a temporary one, the pyrexia increasing whenever the administration of the drug is stopped.

This drug has also the power in some cases of allaying the pains to <sup>a</sup>certain extent.

Phenacetin has a similar effect, and is to be preferred on account of its less depressing action on the heart.

Salol has often been employed, chiefly by those who consider Malta fever to be due to infection of

the alimentary tract. Being composed of Salicylic acid and phenol, it exerts a powerful antiseptic action, but its use does not affect the course of the fever in any way.

Thymol has recently been employed a good deal, and some observers are of the opinion that it has a more beneficial effect than any of the other drugs, but the action is by no means specific.

Bismuth is much employed to relieve the gastrointestinal irritability and has proved very useful, the appetite and general condition improving as the irritability is allayed.

Liq. Hydrarg. Perchlor. is believed by some to act beneficially and Dr Phillips of Cairo has had several good results from its use, but in my own and other cases which I have had access to, the effect was practically nil.

#### Diet:

In superintending the patient's diet much care has to be exercised. Many physicians are inclined to allow a liberal diet long before the patient is in a fit state to digest it.

During the early and acute stages, the diet should consist almost entirely of milk, brandy being added if the cardiac apparatus requires stimulation.



When the fever subsides somewhat, a little beef or chicken tea may be allowed, and later on some light milk food, such as Benger's food, arrowroot, rice or cornflour. The state of the tongue should be carefully watched, and if increased diet is accompanied by a dirtier tongue, then we must retrace a step or two and return to lighter nourishment. Fish, chicken and butcher meat should not be allowed until the patient is well on the road to recovery, with a temperature reaching normal and remaining there more or less, for a fortnight. The dieting in Malta fever requires almost as much care bestowed on it as in enteric fever. The patient's appetite may be much greater than his power of digestion warrants, so that we must not rely too much on the subjective sensations. It is safe to say that the power of digestion, is, as a rule, inversely proportionate to the severity of the symptoms.

In some cases, the nausea is so great that it is only with great difficulty that the patient can be persuaded to take anything, and some doctors make the mistake of saying that such cases should just be forced to eat, and made to take fairly substantial meals against their wills, but it is much safer to rely on the patient's inclinations and give very light diet. In cases with much gastric irritability, and sometimes persistent vomiting, a

little iced champagne may be all that can be kept down, but it may tide the patient over the dangerous period.

With so much milk food, we must avoid the chance of any scorbutic symptoms by giving a little lime juice or the juice of a fresh lemon from time to time. When the patient's condition is very low, a little brandy administered in egg flip, commencing with 2 ozs. per day, is often very useful. Raw beef juice or chicken jelly are both appetising and nourishing. The beef teas and <sup>Meat</sup> Extracts are appetising and stimulating, but not nourishing. Calves' foot jelly, although not nourishing, is pleasant and refreshing.

Crusts of bread, potatoes and oatmeal should be withheld until convalescence is established.

It is easier to regulate the diet in convalescence <sup>from</sup> this fever than in enteric, as the appetite is not so ravenous. Tobacco is better left alone, especially if the heart is excitable and weak. During convalescence some light wine such as Burgundy is beneficial, and a quinine and iron tonic may be administered.

The patient should be confined entirely to bed during the acute stage, and as long as the temperature tends to rise above 100°F. or as long as the tongue is furred or arthritic or neuritic complications are present.

It is usually safest to keep the patient in bed until the temperature has remained subnormal for at least ten days, except towards the end of the very tedious and sub-acute cases, when the tongue is pretty clean, the appetite fair and the temperature only rising to 99° or 100°F. in the evening, in which cases the patient may be allowed to get out of bed for a short time every day.

Many relapses are due to the patient being allowed up too soon and many are due to injudicious dieting. Great patience has to be exercised by those in charge of the case.

Once the patient is able to sit up for a little every day, care must be taken to see that he is not exposed to any draught or chill, and that he is not allowed to fatigue himself. As strength returns the patient may be allowed to sit in a wheel-chair, which he can propel himself as he becomes stronger.

As regards the treatment of individual symptoms: When the patient is admitted to Hospital suffering from the fever in an early stage, he should be put to bed, in a bright, clean, well-ventilated room. It is advisable, especially if any joint troubles are present, to place him between blankets instead of sheets, and in all cases flannel nightdresses or pyjamas should be worn. Several changes of bed-clothes and nightdresses will be required for

changing, when perspiration is profuse, and a mosquito-net should be supplied.

The state of the bowels must be enquired into, and any constipation relieved. For this, castor oil, calomel or an enema may be employed. Should diarrhoea be present, enquire into the amount and nature of the nourishment and restrict it, and it may be advisable to administer some bismuth and opium, or if the tongue is foul and tympanites present, we may give grey powder (gr. I) in a little milk two or three times a day.

If the diarrhoea is very persistent an enema of starch and opium should be administered. The mouth, gums and teeth must be attended to, and for this glycerine and borax, and a chlorate of potash mouth wash are useful.

The surface of the body should be sponged once or twice daily with tepid water, and after being thoroughly dried, the parts exposed to pressure should be rubbed with methylated spirits, and a little starch and boracic dusting powder applied, to keep the skin in as good condition as possible, and so minimise the risk of bed-sores forming.

Should the skin be very dry and hot, a diaphoretic mixture may be given occasionally to produce a gentle perspiration.

More often discomfort arises from too profuse perspiration. This occurs usually towards night, just after the temperature falls. The sweat often literally runs off the patient, soaking pillows and bed-clothes in a minute or two. This profuse perspiration is beneficial except in very debilitated subjects, and only in these should any attempt be made to check it. Great care must be taken to prevent the patient catching a chill, and everything the least damp should be removed at once and dry things substituted - this may entail the use of three or four nightshirts within an hour.

The painful symptoms referable to the joints and nerves are often very intractable. When the pain is acute, it is best allayed by the application of cloths rung out of hot water, or hot poultices. After this the part may be gently massaged with some soothing liniment such as equal parts of Liq. Extract of Belladonna and Glycerine, or equal parts of Lin. Camph. Co. and Lin. Belladonnae.

Warm flannel clothing should be worn next the skin, and if the condition tends to become chronic, Scott's dressing, blisters, electricity and warm baths may be employed.

As the patient's condition improves these symptoms will usually also disappear, as the



neuritis in particular is largely due to the anaemic state of the blood. This may be improved by the administration of Blaud's Pills or other iron tonic - combined with arsenic if the stomach is not in an irritable condition.

Hyperpyrexia is one of the gravest complications and usually precedes a fatal issue. Of course much depends on the idiosyncrasy of the patient. 104°F. means a very serious state of affairs in one patient, while in another patient 104°F. may be attended with little, if any, discomfort.

Hughes<sup>(13)</sup> considers that 103°F. should be considered the border line - anything above 103°F. being more or less serious.

He advises the application of cold to bring down the temperature, wherever it exceeds 103°F.

There are various methods of applying cold, perhaps the best being by sponging with tepid or ice water according to the susceptibility of the patient. Cold packs and ice packs are also employed by some, but cold baths should not be resorted to.

The application should be continued until the temperature falls to 102° or 101°F. but only till then. The temperature should be taken every three or four minutes during the application, and cold should only be applied to the arms, legs and abdomen,



the chest being left alone on account of the risk  
(13)  
of pneumonia.

Any signs of heart failure and pulmonary embarrassment must be carefully looked for, and digitalis and strychnine administered when called for. The headache and general feeling of uneasiness at the commencement of an attack is best relieved by full doses of Potassium Bromide and Morphia, and later on if sleeplessness is a troublesome symptom sulphonal or trional may be administered.

The orchitis is best relieved by the application of cooling, evaporating lotions, or a lead and opium fomentation, and the scrotum should be enveloped in cotton wool and elevated.

Next: The question of invaliding the patient home arises in those cases which occur in sub-tropical climates:

Be careful that the case is really one of Malta fever, and not one of enteric, before you order the patient home in early convalescence. If a patient has been ill throughout the summer months and has recovered in the autumn, it is often advisable to keep them in the Mediterranean, as the weather becomes very pleasant and bracing at this time, whereas in England the patient would just arrive in time to experience all the trials and discomforts of an English winter.

Should the patient recover in the spring or just before the hot season, he is much better in England, as he will find the English summer very beneficial, but care must be taken to see that he is sufficiently well to undergo all the hardships and fatigue which travelling and a sea-voyage entail. A dry bracing climate with plenty mountain air is best suited to such cases, and in this country the Highlands of Scotland afford splendid chances to recuperate. Two towns we would particularly recommend for this purpose, viz. Grantown-on-Spey and Braemar. I have known a man suffering from the effects of Malta fever being taken to Braemar in a stretcher and wheeled about in a bath chair to begin with, and yet be able to walk four miles at the end of a month.

While discussing the treatment of Malta fever, we must not omit to mention the Malta fever Anti-toxin serum. Several successful cases have been reported within the past few years, and no doubt it will be more and more largely used as time goes on. In fact it is to the elaboration of the serum treatment that we most look for reliable results in the future.

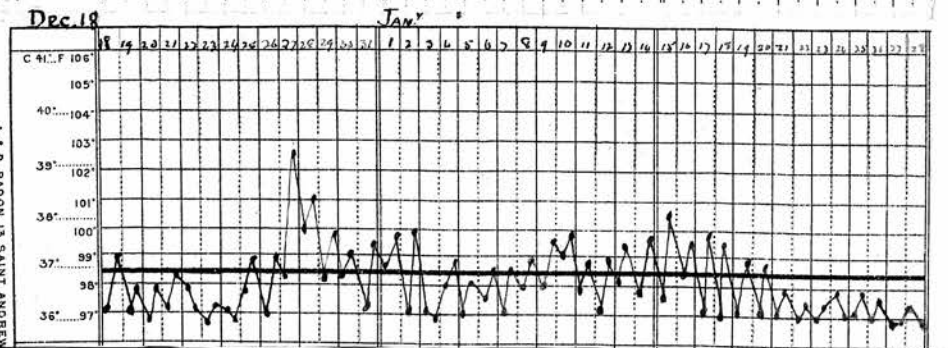
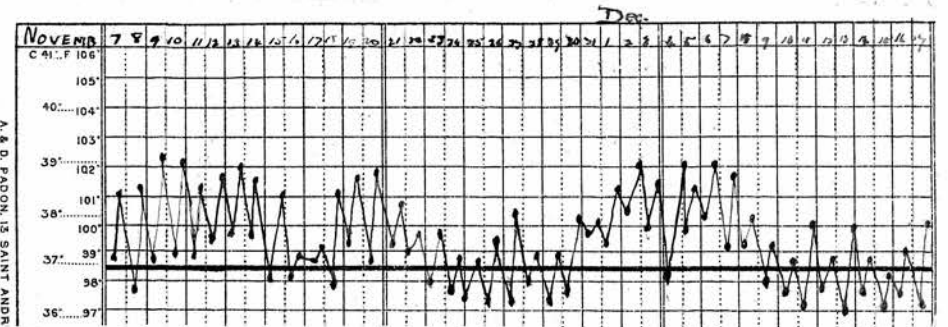
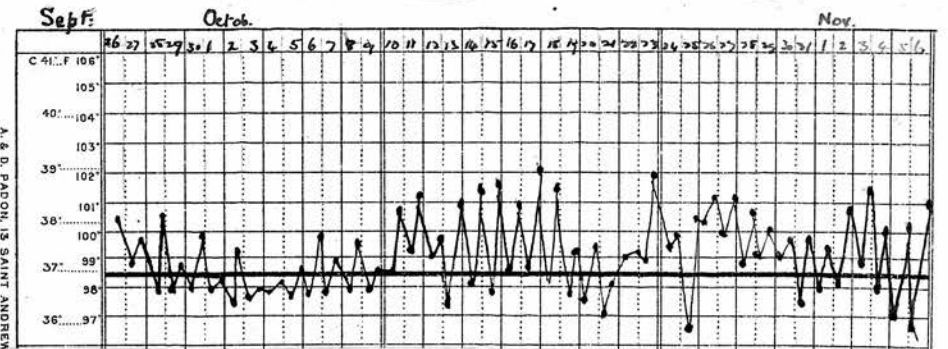
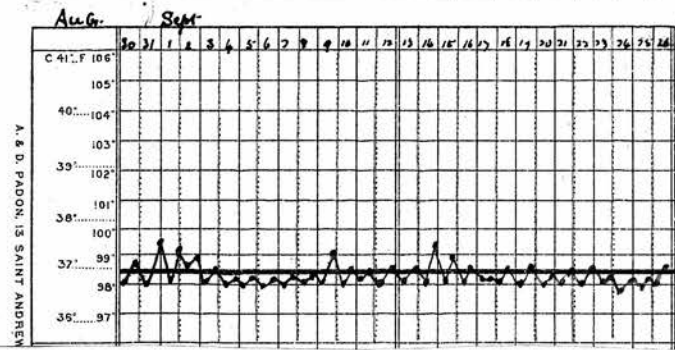
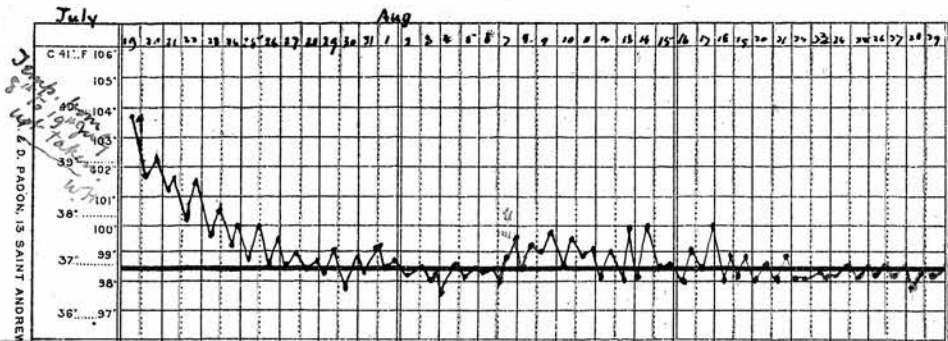
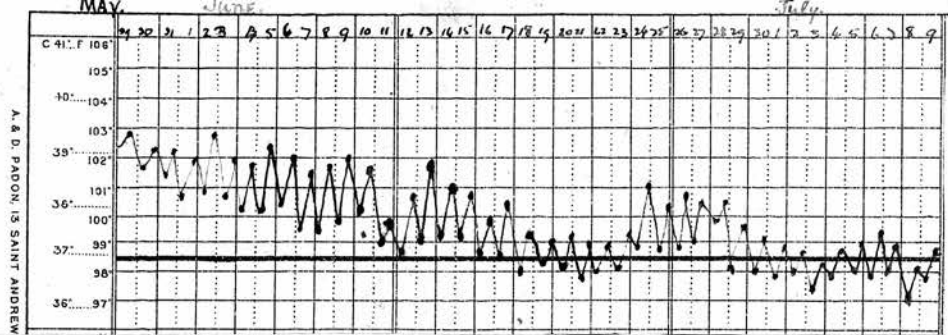
Fitzgerald & Ewart were amongst the first to point out the value of this treatment. They record the use of the antitoxin in a case which

other resisted treatment. (28) A twenty c.c. dose of the serum, thrice given, was sufficient to cure the patient. The third dose which was given a week after the treatment was begun caused high temperature (104.6°F), and the patient became seriously ill with rapid respiration, blood-tinged expectoration, painful swelling of the joints and urticaria. These symptoms soon disappeared, however, and patient made a good recovery.

Staff Surg. Reid records very successful results (29) from inoculation of Malta fever antitoxin - a rapid lowering of temperature almost invariably occurring after a few injections.

It is advisable to combine this treatment with the periodic estimation of the opsonic index. The injection of the antitoxin is found to markedly raise the opsonic index, and additional doses should be given when the opsonic index is discovered to be falling - apart from the usual "negative phase".

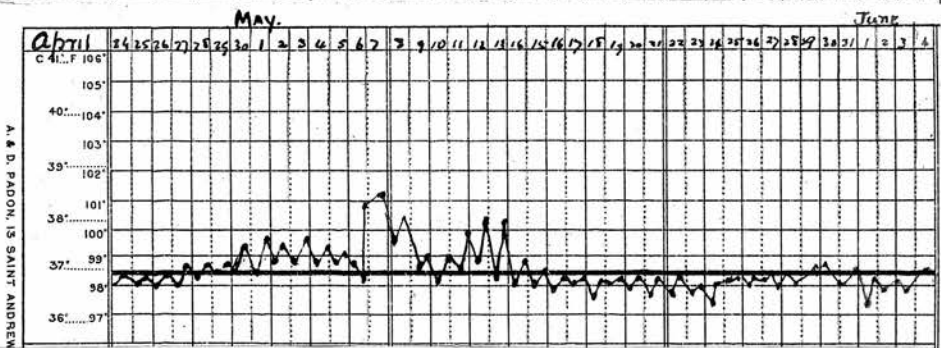
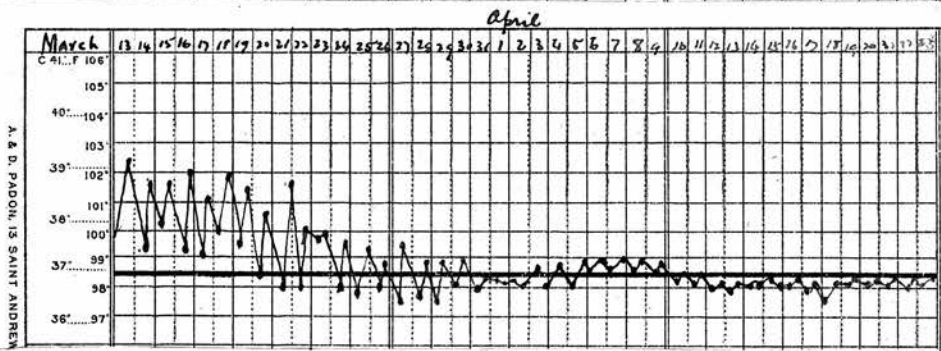
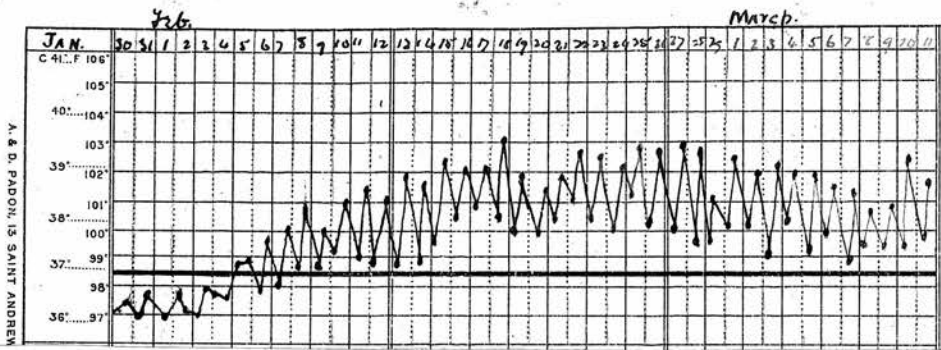
We have already expressed the belief that the antitoxin will prove useful as a prophylactic agent in the future.



My case: representing the Undulatory type:-

My own case is an unusually interesting one, on account of the fact that it was immediately preceded by, if it did not actually run concurrently with an attack of enteric fever.

Returning home from Bombay in the capacity of Ship's Surgeon in May 1905, I began to feel out of sorts and troubled with headaches on the day previous to our entering the Suez canal - about twelve days from the date of our leaving Bombay. The following day I was somewhat worse and complained of nausea, headache and pains in the back of the neck. Believing that I was just suffering from a "touch of the sun", I tried to keep going, but every day found me worse, the pains increasing in severity, and the nausea being so marked that milk and soda water was my sole diet, and even that I took with difficulty. When we reached Algiers - seven days from the date of my first symptoms, I was able to crawl about but not more, and the following day I had to give in and take to my bed. On arriving at Gibraltar two days later, I was conveyed to the Civil Hospital there. Up till this time I had been getting slowly but progressively worse. I felt absolutely miserable, shunning all society, and was constantly experiencing creepy and shivery





sensations: I would be cold one minute and hot the next, but during the later days I was flushed and feverish, and my appetite had entirely disappeared. Tongue was covered with a thick fur, and was swollen and indented laterally by the teeth; I had a bad taste in the mouth, bowels were constipated, and I lost flesh rapidly. The two occasions on which I took my temperature on board ship the thermometer registered about 102.5°F.

Admitted to Gibraltar Hospital on May 31st.

On arrival at Hospital was in a somewhat collapsed state: temperature 102.2°F. Pulse 100 per minute.

General appearance: Pallor and considerable emaciation. Expression pained and anxious - eyes congested - tongue coated with a thick yellowish white fur - lips and teeth covered with sordes.

Heart's action - weak and excitable.

Tenderness in epigastrium. No tumidity of abdomen, but two or three small spots in hypogastric region, <sup>and</sup> occasional gurgling in iliac fossae.

Lungs - healthy.

Kidneys - urine acid, high-coloured and scanty: deposit of urates.

S. G. = 1028.

Complaint:

Headache and pain behind the eyes. Pain in the back.

Feeling of exhaustion.

Slight epistaxis, and nasal and pharyngeal catarrh.

Loss of appetite, nausea and some abdominal discomfort.

Course:-

When admitted I was confined to bed, and diet restricted to milk and potash.

Calomel gr.II. was administered.

Was very restless and slept very little the first three nights. Bowels moved on the second day, but fever did not abate.

Trional gr.XX. taken on the fourth day resulted in sound sleep.

Diarrhoea started on third day, and persisted for the next three weeks, the stools early assuming the "pea-soup" character associated with typhoid. As a rule only one copious motion per diem, but occasionally two.

My case was diagnosed as one of enteric fever, and treated accordingly.

My temperature was of the remittent type, the morning reading being about one and a half degrees lower than the evening, but never falling below 100°F until the 7th June - ten days after admission,

when it registered 99.4°F. The highest readings at night were 102.6°F on the 2nd June, and 102.5°F. on the 5th June. From 7th June my temperature remained below 102°F. both morning and evening.

The evening rise reached its height between 8 and 9 p.m. as a rule, and then rapidly fell, the fall being succeeded by a period of profuse perspiration which continued till close on midnight.

The temperature curve came down to normal, for the first time, on the 18th June, and remained at or about normal until the 24th, when it rose in the evening to 100.4°F. It continued to go up in the evening for the next five days and then settled again, and remained at or about normal, but for an occasional slight rise in the fifth week, when I was getting out of bed for a short time daily.

The fever left me in a very weak and emaciated condition, but shortly after my temperature became normal, improvement was extremely rapid and my appetite was ravenous.

Although my case could not be described as a severe one, yet I had one or two unpleasant complications.

At the end of the third week, reckoning from the time of my admission into Hospital, I was much troubled with severe neuralgic pains in the lower

limbs, and some fullness and swelling of the right knee-joint. There was also a sensation of numbness and tingling in the skin of the thighs above both knees.

About the end of the third week, I suffered from a sharp, though fortunately short, attack of urethritis, which was attended with the usual painful and distressing symptoms of that condition. The urethritis, together with the discharge soon disappeared, but left a somewhat swollen and tender vas deferens and epididymis on the right side.

On the 7th June - about seventeen days from the onset of first symptoms, my blood was tested for the serum reaction both to typhoid and Malta fevers, but it was negative to both in a dilution of 1 in 10.

On the 15th June - on the test being again applied, the blood reacted slowly to Typhoid fever in a dilution of 1 in 20 but not at all to Malta fever.

Blood count on the 12th June:-

Red Blood corpuscles	4,000,000	per c.m.
White " " "	11,000	" "

The notes of my case, which I have just given, are somewhat scanty, owing to the fact that the Hospital record of my case, which furnished me with material from which to make my notes, was found to be anything but complete.

Before leaving Gibraltar, which I did six weeks from the date of arrival, I had returned to ordinary diet and was relishing it, but I had scarcely been two days on boardship, on my way home, when my appetite began to fail, and I began to feel not quite up to the mark.

This was soon followed by nausea, headache, pains behind the eyes, and creepy sensations. I could not keep warm as long as I continued to go about, so took to my bunk eventually.

My appetite soon disappeared entirely, tongue became thickly furred and bowels were constipated. I confined myself to milk and soda and took a dose of castor oil but the latter only afforded very slight and temporary relief. By the time we reached London I was very feverish and extremely weak and naturally thought that I was suffering from a relapse of typhoid fever. Although it was somewhat risky I decided to travel straight on to Edinburgh, and on arrival there felt even more ill and miserable, and went straight to bed when I got home.

Temperature then registered 103.8°F. and pulse 104 per minute.

The following day - July 19th - I was transferred to the typhoid ward of the Fever Hospital at Colinton Mains.

State on admission:

Severe headache: nausea and desire to vomit: pain and tenderness on epigastrium: abdomen distended and suggestive of typhoid: nasal catarrh and epistaxis with marked congestion of head. Markedly jaundiced, and scanty bile-stained urine which was found to contain a trace of albumin.

Temperature - 103.5°F.

Pulse - 94 per minute.

Course:

The lower bowel was irrigated which afforded some relief, and on the morning of the following day my temperature fell to 101.8°F.

I was confined to a diet of milk, and chicken tea; bowel was irrigated and I was sponged night and morning with tepid water.

Temperature continued to fall, but I was very restless and somewhat delirious at night for some days.

After eight days my temperature was normal and I felt much better, my bowels at this time tended to be constipated and at no time had the stools the appearance of typhoid stools.

As my temperature continued to remain down, I was put on light ordinary diet very soon, but had no relish for food, and often loathed it. During this attack my pulse was never above 100 per minute as long as I remained in bed, and after the temper-



ature returned to normal and subnormal, the pulse rate was only from 44 to 65 per minute for several days.

Although I remained free from fever, improvement was so slow as to be hardly noticeable, and the most unpleasant symptom was the nausea and complete loss of appetite, with much pain and discomfort in the stomach, after even the very lightest food. Stomachic tonics and sedatives were taken without the least benefit being derived.

On August 5th I ~~was~~ allowed to get up for a little, but was confined to bed again on the 7th, owing to a slight return of the fever and a very rapid pulse with palpitation. Pulse rate was as high as 122 per minute.

During the following ten or eleven days my temperature gradually returned to normal and palpitation disappeared. The discomfort after food and nausea continued however, and at times the pain in the epigastric region was very severe - especially about  $1\frac{1}{2}$  hours after food.

Bowels continued to be constipated and stools frequently contained long strings of mucus at this period of my illness. Although still very weak I was not long in getting about again, but being very thin, I felt the cold very much, and however much food I took, I failed to put on weight.

One day when dressing I was seized with a severe pain in the right hip which caused me to limp when walking. I tried to work it off, thinking I had strained a muscle in some way, but it got rapidly worse and within three hours from my first experiencing it, it became so severe as to make me powerless to move. It was with much difficulty that I got back to bed again, but once in bed and lying perfectly still the pain subsided somewhat - the least movement <sup>however</sup> caused excruciating pain. There was tenderness on pressure over the right sacro-sciatic notch, and over the right sacro-iliae joint, and at times there were all the signs and symptoms of acute sacro-iliac disease; soon the pain extended to the interior of the pelvis - more particularly affecting the rectum and muscular floor of the pelvis.

The pain was at times agonising, and the slightest movement was enough to set up a paroxysm of pain.

Defaecation, coughing or sneezing were accompanied by almost unbearable pain, and I would often allow my bowels to go unrelieved for days, in order to avoid the pain which a motion entailed. The doctor in charge of my case did not appear to understand the condition at all, and seemed to think I was simply suffering from some hysterical condition.

I myself considered it to be some form of neuritis, as it was, above all, a nerve pain. No medicine had the slightest effect in allaying the pain. My appetite continued poor and I had occasional slight rises of temperature while confined to bed with this pain.

In addition to the above I had several painful swellings over the left femur and both tibiae. These node-like swellings were very painful - especially at night and extremely tender to the touch, but were quite transient, only lasting a few days at one spot, and being replaced by others elsewhere.

The Widal test for typhoid fever was applied to my blood on the day following admission and the result was positive, in a dilution of 1 in 20.

The agglutination reaction for Malta Fever was not employed. As the neuritis showed no signs of abating, I was removed to the Royal Infirmary to be under the care of Dr James, for the purpose of receiving application of the high frequency current. When admitted to the Infirmary on September 26th my temperature was found to be higher than it had been for some time previously, viz. 100.2°F and pulse 103 per minute. Temperature swung daily - about two degrees separating the morning and evening temperatures, and it was of the intermittent type, always falling below normal every morning. The temperature

remained below 102°F until October 17th when the evening reading was 102.2°F. It remained about normal from the 19th till the 22nd, then became remittent and intermittent till the 16th November, when it came down to normal and remained there for two days, only again to be followed by another wave of pyrexia which persisted until November 24th.

Still another wave occurred in the beginning of December, and lasted about a fortnight.

Then ensued a period of apyrexia which lasted eight or nine days followed by a much shorter wave.

During the first half of January the temperature kept swinging and did not remain subnormal until the 22nd, from which date until 4th February it never once left the subnormal level.

On the 4th the temperature went up in the evening to 98.6°F. and on the 5th to 98.8°F, when I was advised to return home.

During that weary five months I suffered from all the discomforts and complications which are so typical of the undulatory type of Malta fever.

When admitted to the Infirmary, the following notes were taken:

State on admission: Patient has lost much in weight and looks very depressed and anxious.

Alimentary System: Tongue furred, nausea and loss of appetite and epigastric tenderness. Bowels

require medicine every other day.

Abdomen - no tumidity, no gurgling, liver and spleen not enlarged to any extent.

Rectal examination: Pain on right side on pressing finger against the pelvic wall.

Circulatory system: Pulse varies in rapidity, and is of low tension.

Haemopoetic system:

Hb	90%
R.B.C.	3,800,000
W.B.C.	12,000

Blood film showed no distinctive characters.

Nervous system: Pain and tenderness over a sacro-sciatic notch. Special senses unaffected.

Locomotor system; No visible change in any joints, pain at sacro-iliac joint on moving leg.

Urine: Bright amber colour.

Spec. Grav. = 1020

No abnormal constit.

Blood examination: by T. Shennan on Dec.5th.

Reactions: No reaction with B. Typhosus.

" " " B. Paracolon.

" " " B. B. Paratyphoid.

Marked reaction with micrococcus Melitensis.

December 10th:

Marked reaction with micrococcus Melitensis with 1 in 10 and 1 in 50 dilutions. Not very definite with 1 in 100.



December 20th:

Strong reaction with micrococcus Melitensis in dilution of 1 in 20; 1 in 40; 1 in 60; 1 in 100; and 1 in 200 in 20 minutes.

During the first few weeks in the Infirmary my chief complaint - apart from the pyrexia - was the severe neuritic pains in my right hip; these were at times almost unbearable, and the least movement caused marked aggravation of the pain.

On the 27th September - the day after going to the Infirmary - I was treated with the high frequency current and again on the 28th but without any benefit. As my temperature continued to remain up, I was confined to bed and for the next few weeks suffered very much from pain in the hip and within the pelvis - the latter was especially severe when my bowels moved and set up reflex contraction of the sphincter and muscles which frequently prevented any motion being passed. Dr Gulland made a rectal examination and thought he felt some swelling on the right side, and as my leucocyte count was 12,000 per c.m. he was inclined to advise operation, as he thought possibly some inflammatory matter had formed. Mr Wallace and Dr James, however, thought it was purely a nervous condition and failed to make out any definite swelling. As it turned out, they were correct, the painful symptoms disappearing



suddenly after having persisted for six weeks. A few days later the same symptoms occurred in the left hip and were quite as severe, but they disappeared from the left side after five weeks duration.

The next complication occurred about the same time, and was of the nature of an acute orchitis and epididymitis of the right testicle. The scrotum swelled up to the size of a large orange, the skin over it was tense, red and glazed and the condition gave rise to much suffering, before it resolved about fourteen days later. Soothing applications were applied and the scrotum elevated.

The next complication was an acute arthritis of both shoulders which rendered any movement at these joints impossible. There was slight swelling of the joints, but little heat and no redness over them.

When the acute stage was over the swelling disappeared, but some stiffness with impairment of movement persisted until convalescence was established some four or five months later.

Nausea and loss of appetite persisted, but I was forced to take ordinary food which led to severe epigastric pain after meals, and great gastric distress.

As long as the pyrexia persisted I was much troubled, every forenoon, with creepy, shivery

sensations up my back, which were replaced in the afternoon by a feeling of great heat and thirst. The face became flushed and hectic looking, and there was a burning heat. This condition reached a climax every day about five or six o'clock when the temperature began to subside and by 8 p.m. or 9 p.m. a profuse diaphoresis usually occurred, which usually necessitated my nightdress and bed-clothes being changed three or four times within the course of an hour or two.

By 11 p.m. I was usually rather cold and collapsed. I also noticed that excitement or emotion, such as occurred from an unexpected visitor, resulted in a profuse perspiration.

Insomnia was also a feature of my trouble from time to time, and, when very persistent, recourse had to be had to sleeping draughts, such as trional. Gastric discomfort and constipation persisted throughout my stay in the Infirmary.

I got out of bed for a few hours daily, from the 27th January, until I left the Hospital, but my temperature went slightly above normal on the two evenings previous to my departure. This rather suggested the possibility of the commencement of another wave of pyrexia and it would probably have been safer to have deferred my removal for some days, but the Infirmary authorities considered I had been

long enough on their hands and advised my discharge.

It is more than probable that the comparatively long period of subnormal temperature in the latter part of January, and first few days of February, might have been the commencement of a satisfactory and permanent convalescence, had I been carefully treated as regards diet and rest, but I was encouraged to eat everything that was going - and some infirmity dinners require some digesting! - and was told to walk about as much as possible so as to strengthen myself! . The consequence was that my stomach got quite upset and I overtaxed my strength, and the removal home on a very cold spring day was sufficient to bring back the fever in more than its former severity. During the first few weeks of my residence at home, my condition was decidedly serious. My temperature assumed the remittent type, frequently rising to between 102°F and 103°F, and the constitutional disturbance was very great. I was extremely restless, delirious at night, was distinctly jaundiced, tongue was dry and brown, and thirst was great. Had extreme tenderness in epigastric and hepatic regions, and much pain in the region of the liver. Urine was scanty, high-colored, bile stained and contained albumin. Diarrhoea with very offensive stools and great nausea were two of the chief symptoms. When my

condition became somewhat less acute I developed additional symptoms, such as deafness, and noises in the head, hyperaesthesia of the feet and toes, and temporary loss of voice, with acute pain in larynx when I spoke or swallowed.

These symptoms came on as the temperature declined, and when it reached normal about the 20th March, severe joint pains were developed at the same time. On this occasion it was the smaller joints which were affected - chiefly the wrists and fingers, which suddenly swelled up and became red and hot, and exquisitely painful and tender. The left shoulder at the same time became worse again, although not so bad as on the previous occasion. From the 20th of March until the end of that month the temperature was of the intermittent type, then followed a long period of normal and subnormal temperature throughout April, with a final brief period of slight pyrexia about the middle of May, after which I entered on a slow convalescence.

During April, when my temperature remained normal or subnormal, the painful complications gradually subsided, the nausea disappeared, and my tongue began to get thoroughly clean and lost its angry red tip for the first time. The only new complication at this time was a swelling of the thyroid gland, which gave rise to only slight discomfort, and



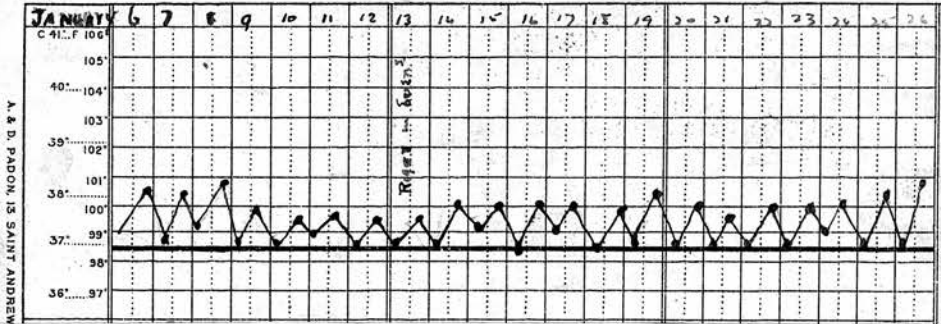
soon subsided. My pulse rarely went above 100 per minute except when I suffered from over-fatigue or excitement, when palpitation was frequently present and produced on the slightest provocation.

When convalescence commenced I was extremely emaciated and naturally very weak, and when going about noticed a distinct paresis of the extensor muscles of the leg which produced a mild condition of drop foot. There was also some swelling of, and pain in, the testicle and cord, which had previously been the seat of acute inflammation.

When recuperating at Braemar I had a slight relapse in the month of August, which lasted 12 days and which I distinctly traced to over-exertion as it followed on my climbing a hill 1500 feet high. Since that relapse I have steadily improved and am now almost back to my original weight, notwithstanding the fact that I spent the latter months of the year in reading for a pretty stiff examination, which I sat for in January.

The only weakness which is now present is in the intestinal tract, as I still notice some mucus and streaks of blood in the stools if I indulge in a too liberal diet or expose myself to chills, but that is becoming less and less frequent.

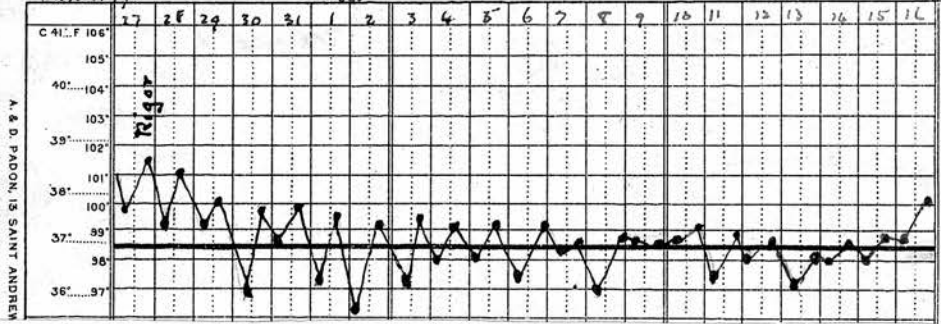
D.P.



January

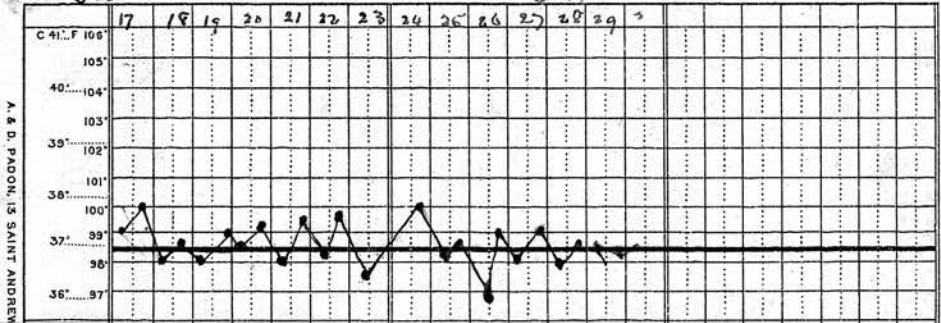
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D.P.





The second case which I wish to draw attention to is that of D.P. which represents the ambulatory type.

Dr P. began to feel ill about the middle of December 1906.

To begin with he just felt out of sorts, and had vague aches and pains in various parts of his body. A persistent headache and nasal catarrh were the next symptoms to arise, and he began to feel chilly and inclined to linger near the fire. He then suffered from loss of appetite, constipation and a certain degree of nausea, but continued to perform his hospital duties until about three weeks had elapsed from the time he first fell ill.

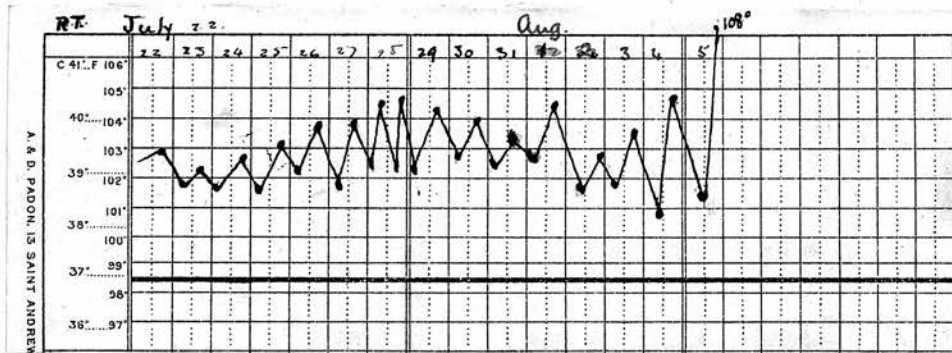
On January 6th - nineteen days from the date of onset of symptoms, he began to suspect that he might be suffering from more than a "touch of influenza", and sent his blood to be examined for the agglutination reaction. The following day he was informed that his serum reacted to the micrococcus *Melitensis* markedly in a dilution of 1 in 20, and slowly in 1 to 40. His temperature at this time was 100.2°F. in the evening, and normal in the morning. It continued to be of the intermittent type for over a fortnight - usually reaching 100°F. Every evening, but in the third week it was slightly remittent and went up to 101°F, it then gradually

settled down to normal and remained normal and sub-normal until the end of sixth week, when the temperature again became slightly irregular, but never rising above 100°F. The pyrexial condition entirely disappeared within two and a half months from the onset of symptoms.

Dr P. continued to go about throughout the attack, and the only unpleasant symptoms for most of the time were the headache, nausea and loss of appetite with furred tongue, and constipation. On two occasions he had a rigor, with which was associated a more severe headache than usual. The first rigor occurred on the evening of January 13th - the 26th day of his illness, and the second took place on January 27th - the 42nd day of illness.

He took no medicine at first, but from the 30th January he took thymol gr.V. thrice daily and from that time the temperature came rapidly down. He had confined himself to a milk diet while suffering from pyrexia, but on 5th February he returned to ordinary diet, with the exception of meat which was excluded.

The agglutination test was applied a second time towards the end of January and it was found that clumping of the micrococcus Melitensis took place in as high a dilution as 1 in 200.



During the final stage of the fever, when the temperature tended to become somewhat irregular, Dr P. complained of vague joint pains which persisted for about a week. When the temperature had finally returned to normal, the serum test was again applied and gave a positive result, but only in a dilution of 1 in 10 and that only after three hours. Dr P. continued to take thymol for some time after convalescence was established.

The Malignant type may now be briefly dealt with.

Owing to the very low mortality rate of Malta fever, such cases are naturally but rarely met with, but I shall present a case, the records of which I have had the opportunity of studying.

This was the case of R.T., a strong, healthy young soldier, aged 24, who was somewhat suddenly seized with severe pain in the head, together with sickness and some vomiting. He soon became very feverish and suffered from severe constitutional disturbance. His tongue was dry and brown, he was slightly jaundiced, and had marked tenderness in the epigastric region.

Diarrhoea occurred on the second day of the fever and persisted throughout the illness. The motions were very offensive and slightly blood stained.

Pulse was full and bounding. Respirations were somewhat rapid and the breath sounds were accompanied by a few râles and rhonchi, and in a few days there was evidence of oedema at the bases of the lung.

Urine was scanty, high-coloured, spec. grav. 1.030 trace of albumin.

Patient was troubled with sleeplessness and was delirious at night.

The temperature was 102.8° F. when first taken - at 3 p.m. - and the same night it went up to 103.5°F.

It remained constantly up for the next few days, keeping as a rule in the region of 104° and 105°F. It then dropped to 101°F and remained down for two days, then rose suddenly to 108°F at which point patient died, death being due to heart failure resulting from the hyperpyrexia. No joint or nerve complications of an inflammatory nature were present.

Comments on above cases:

My own case furnishes one of the most typical illustrations of the course and complications of an ordinary severe attack of Malta Fever, embracing as it does, almost, if not quite, every phase of the disease.

Its duration is somewhat doubtful, as it is quite possible that I was suffering from Malta Fever at the same time that I was in the throes of enteric fever.

No doubt the initial symptoms were due to typhoid alone, although Dr James of Edinburgh and others who studied my case went the length of saying that they considered that I had been suffering from Malta Fever from the commencement, and that enteric fever was at no time present. This view, I think, is very unlikely to be the correct one. Because, if the early symptoms, which came on while we were in the Red Sea, were due to Malta Fever, it would mean that I received the infection while at Bombay, as there was no chance of being infected while on the voyage - neither mosquitoes, goat's milk nor any other agent being present, which would be at all likely to convey the disease.

I grant that the incubation period, as calculated from the day of departure from Bombay, was of a duration which rendered either Malta fever or



typhoid fever possible, and we know that it is held that Malta fever does occur in India, but I never came in contact with, or heard of a case occurring in Bombay. Besides, another reason for my thinking that enteric was the disease first present, was the fact that a steward on board ship developed a very typical attack of enteric about the same time as I did, and ultimately died of the disease at the English Hospital at Algiers. His case and my own I attribute to some infected cow's milk which we partook of at Bombay.

The initial symptoms in my case might have been due either to the Malta fever, or to enteric at its prodromal stage, but the temperature curve, the pea-soup stools and the positive result of the Vidal reaction point to enteric although there was no tumidity of abdomen, gurgling in iliac fossae, or rash on abdomen at this period.

The epididymitis which occurred was less likely to be the result of Malta fever, than of the urethritis which, in turn, was probably due to typhoid infection. The joint pains, neuralgia and sweatings which occurred about the end of the third week of my stay in Gibraltar point rather to Malta fever, and the short period of pyrexia which occurred in the fourth week, and which was at the time considered

to be a relapse of enteric fever, might be taken along with the last-named symptoms, as representing the first evidence of Malta Fever.

It was mentioned, when going over my case, that the first occasion on which the serum reaction was ascertained it was found to be negative both to typhoid fever and malta fever. The only way in which I can account for this, when there was such marked clinical evidence that enteric existed, is by supposing that the agglutinins of both typhoid and Malta fever were present, and that they destroyed each other, as Dr Phillips<sup>(19)</sup> and other observers have pointed out does occur. If that was the condition of affairs in this instance, the agglutinins of the typhoid infection evidently soon got the upper hand as the Widal test was positive a week later. If I really did suffer from Malta Fever at this early period, the infection might have been contracted either at Algiers, where we spent two days, or in Gibraltar Hospital. This introduces the question of "mode of infection".

Personally, I am strongly inclined to favour the mosquito theory, and that theory would hold whether I was infected at Algiers or at Gibraltar. At Algiers I received several severe mosquito bites and on the other hand, only took the ship's supply of condensed milk, never touching either goat's or

fresh cow's milk. In fact, I had no food on shore at that port except one or two cups of café noir.

If, on the other hand, the disease was contracted in Gibraltar - as it must have been if the fever only commenced after leaving that town - the mosquito theory could again very well hold. In the first place there was a case of Malta Fever being treated in the ward I was in, which, together with the fact that mosquitoes were plentiful, and that I had no mosquito curtain, appear to me to provide all that was necessary to produce the infection.

In the second place there were two sisters on duty in this ward at the time, and they, as well as myself, complained on more than one occasion, of being bitten by mosquitoes. These two sisters both contracted the fever that summer, while the sisters on duty in the wards which contained no Malta fever cases, all escaped.

Regarding the question of goat's milk as an agent in transmitting infection - we now know that it frequently contains the specific organism and that animals fed on artificially infected goat's milk do develop the disease, so that the possibility is that goat's milk is a factor, but I do not consider it to be by any means the most important one. Personally, I was given only cow's milk and all milk was sterilised, which in my opinion precludes the

possibility of goat's milk being a factor in the causation so far as my case is concerned.

Dr P., on the other hand, says that his attack was traced by Major Horrocks to some cows who supplied the hospital with milk.

He said the milk was supposed to be boiled, but thinks the boiling must have been omitted - one way, certainly, of explaining away things.

Regarding the question of <sup>bad</sup> Sanitation being concerned in the causation, of the cases which occurred in Gibraltar Hospital, I may state that the drains had been recently laid and were in good working order and all the sanitary arrangements were thoroughly up to date.

Personally, I think that I only began to suffer from Malta fever on the voyage home from Gibraltar. It was only then, that I began to experience regularly the creepy sensations and profuse perspirations which later on formed such characteristic symptoms.

It was rather strange that my abdomen had the full and distended appearance only after returning to Edinburgh, at which time I was much more probably suffering from Malta fever than typhoid.

Looking back on the first few weeks following my return to Edinburgh, it seems almost incredible that the nature of my symptoms, combined with the fact that I had just returned from the Mediterranean

I omitted to mention that the peculiar mousey odour which has been observed in some cases, was very noticeable in mine; also, that while in Colinton Hospital I developed Pityriasis Versicolor, which persisted well into convalescence. I have not seen it recorded in any other case, but its presence was no doubt due to extreme debility.



did not suggest the possibility of Malta Fever to my medical attendants in Edinburgh. The Widal test for typhoid was carried out and found to be positive, but the serum reaction for Malta Fever was never employed in Edinburgh until I had been suffering for months. The most typical phase of the fever was that which was present while I was a patient in the Royal Infirmary, and subsequently at home.

I think that I had almost, if not quite, all the typical symptoms and complications met with in the disease - nausea, dirty tongue with red tip and edges, epigastric tenderness, creepy sensations before the pyrexia and profuse perspirations after it: palpitation, anaemia, constipation with passage of mucus and traces of blood in stools, joint pains, severe neuritis: sleeplessness, restlessness, numerous relapses and affections of special senses. It might specially be noted that there was never, at any time, anything abnormal in the lungs, and strange to say, the spleen was never enlarged to any appreciable extent, and the liver only for a short time. It is stated that the glands of the body are often enlarged and in my case the thyroid was markedly enlarged, although not an ordinary variety of gland tissue. During the course of the relapse which occurred at



Braemar, some months after, I left my bed, the supra-clavicular group of glands was markedly enlarged and so inflamed that they appeared on the point of suppurating. Another point of interest was the fact that slight albuminuria was present when at Colinton Hospital and at home, which pointed to my case being a severe one. My temperature attained its daily maximum between 3 p.m. and 5 p.m. as a rule, and not towards night, as is frequently the case. The neuritis which was my chief complaint for many weeks, was at first attributed, in part, at any rate, to neurasthenia, and for that reason electrical treatment was recommended. I would advise that in the case of any patient, recently returned from abroad, who is suffering from any form of neuritis, the agglutination test for Malta fever should be applied, and that not till then should the condition be put down to neurasthenia, debility or chronic rheumatism, as the case may be.

I have previously stated that the result of the widal test for typhoid fever, when applied in the Infirmary was negative, which means that by this time the agglutinins of Malta fever had got the upper hand of the typhoid agglutinins. The practical importance of this is that if in such a case, the Widal test for typhoid is alone applied, and the result negative, one might naturally be

inclined to think that the patient was quite free from typhoid and be allowed an unrestricted diet, with serious results, because it might be a case in which both diseases were present, but in which the agglutinins of Malta fever had got the better of the typhoid agglutinins and destroyed them, thus rendering the Widal reaction negative; where the patient has been abroad, it would be advisable always to test for Malta fever in addition to doing the Widal test, and to treat the case as if it were typhoid.

We know that many advise a liberal diet in fevers, but we think that in Malta fever, at any rate, much harm may be done and the disease unnecessarily prolonged as a result of such management. As complete rest as possible both to the body as a whole, and to the digestive organs in particular, we consider most important, also the avoidance of chills and any form of mental excitement.

Dr P's case calls for very little more notice.

Attention may be directed to his having two rigors in the course of his illness. Rigors, we know, are rare, especially in the mild form from which he suffered, but they may have been induced in his case by some chill as he continued to go about although feverish.

Another point of interest is that his agglutination test became markedly weaker as the fever progressed. This, according to Birt and Lamb, is common in the prolonged cases of the undulatory type, but the short mild cases have usually a high and a rising reaction.

I have already stated that Dr P. considered cow's milk to be the source of his infection, as the animals which provided his milk, reacted to the micrococcus Melitensis.

Dr P. had marked relief from the time when he stopped taking meat in any form and confined himself to a light diet.

As regards the treatment of Malta fever by drugs:

My own case illustrates very well the futility of drug treatment in Malta fever.

In the course of my illness, I was given a trial of most of the medicines which have been employed to combat the disease.

The following were given for various periods in the following order:-

(1). Rx Liq. Hydrarg. Perchlor. in V.  
 Liq. Strychnine Hydroch. in V.  
 Liq. aq. ad. - 3 p t.i.d

(2). Rx Salicin gr.II.  
 Phenacetin gr.II.  
 Quin.Sulph.gr.II.

+ i.d

- (3). Phenacetin gr.X + 1.D
- (4). Yellow Santonin, gr.III, B.I.D.
- (5). Sod. Salicylat gr.XII + 1.D
- (6). Antipyrin, gr.X + 1.D
- (7). Aspirin, gr.X B.I.D.
- (8). Quin. Salicylat. dose not stated.
- (9). Quin. Sulph. gr.III+1.D

The above were tried with the hope that one might prove to have some specific effect, and diminish the temperature, but they were all useless. None of the nerve sedatives had any effect in relieving pain, and most of them had a general depressing effect.

The stomach irritability and dyspepsia were treated with bismuth, salol and pepsencia; the heart was strengthened by V. minim doses of Tr. Digitalis t.i.d. and narcotin and trional were given on various occasions, to relieve the sleeplessness.

Dr P. referring to his own case and his experience in treating those under his care, states that he finds Thymol (gr.V. twice or thrice a day) to have the most beneficial effect.

At present everything points to the administration of Malta fever antitoxin being the treatment of the future, and no doubt protective inoculation will play a prominent part in the prophylaxis of Malta Fever. Good work has recently been done and continues to be done in the direction of finding out the chief factors in the etiology of the disease, and once these have been satisfactorily determined on, a great step will have been taken towards the eradication of this distressing malady.



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